

A TEXT-BOOK  
OF X-RAY DIAGNOSIS  
BY BRITISH AUTHORS

EDWARD WING TWINING 1887 1939

THIS VOLUME IS DEDICATED TO THE MEMORY  
OF EDWARD WING TWINING IN GRATEFUL  
APPRECIATION OF THE PART HE TOOK IN THE  
PRODUCTION OF THIS TEXT BOOK AND OF HIS  
OUTSTANDING CONTRIBUTIONS TO RADIOLOGY

# A TEXT-BOOK OF X-RAY DIAGNOSIS

BY BRITISH AUTHORS

IN THREE VOLUMES

Edited by

**S. COCHRANE SHANKS, M.D., F.R.C.P., F.F.R.**  
HONORARY DIRECTOR X-RAY DIAGNOSTIC DEPARTMENT UNIVERSITY COLLEGE HOSPITAL ETC

**PETER KERLEY, M.D., F.R.C.P., F.F.R., D.M.R.E.**  
PHYSICIAN TO THE X-RAY DEPARTMENT WESTMINSTER HOSPITAL  
RADIOLOGIST ROYAL FREE HOSPITAL ETC

AND

**E. W. TWINING, M.R.C.S., M.R.C.P., F.F.R., D.M.R.E.**  
RADIOLOGIST ROYAL INFIRMARY MANCHESTER RADIOLOGIST CHRISTIE HOSPITAL  
LATE HUNTERIAN PROFESSOR, ROYAL COLLEGE OF SURGEONS,  
LECTURER IN RADIOLOGY UNIVERSITY OF MANCHESTER ETC

VOLUME III

With 710 illustrations



LONDON

H. K. LEWIS & Co. Ltd.

1939

*First Edition*  
*Reprinted*  
*Reprinted*

*December 1939*  
*January 1943*  
*February 1944*



*The Typography and Binding  
of this book conform to the  
authorised economy standard*

## PUBLISHERS' NOTE

THE size of the volumes in this reprint has been reduced in order to comply with the regulations now in force prescribing the area of the type surface in relation to the size of the page.

The type area is the same as in the first issue and the reduction in size has taken place at the expense of the margin.

The Publishers regret this reduction, one which somewhat mars the appearance of the volumes, and they hope that it will be accepted as a war time necessity. When a new edition of the work is called for it will be their endeavour to return to the original format.



## LIST OF CONTRIBUTORS

- S. L. BAKER**, Ph.D., M.Sc., M.R.C.S., L.R.C.P., D.P.H.,  
Procter Professor of Pathology and Pathological Anatomy,  
University of Manchester.
- HUGH CAIRNS**, M.A., D.M., B.S., F.R.C.S.,  
Nuffield Professor of Surgery, University of Oxford; Surgeon,  
Neuro-Surgical Department, London Hospital; late Surgeon,  
National Hospital, Queen Square; late Surgeon, Hospital for  
Paralysis and Epilepsy, Maida Vale; late Hunterian Professor,  
Royal College of Surgeons.
- I. CAMPBELL GOLDING**, M.B., Ch.M., M.R.C.P., D.M.R.E.,  
Assistant Radiologist, Middlesex Hospital and British Red Cross  
Clinic for Rheumatism; Honorary Radiologist, Royal National  
Orthopaedic Hospital.
- E. DUFF GRAY**, M.A., M.D., F.F.R., D.M.R.E.,  
Honorary Radiologist, Royal Infirmary and Ancoats Hospital,  
Manchester; Visiting Radiologist, Park Hospital, Davyhulme.
- H. K. GRAHAM HODGSON**, C.F.O., M.B., B.S., F.R.C.P., F.F.R., D.M.R.E.,  
Honorary Physician in charge Department of X-ray Diagnosis,  
Middlesex Hospital; Honorary Radiologist, Central London  
Throat, Nose and Ear Hospital.
- DONALD HUNTER**, M.D., F.R.C.P.,  
Physician, with charge of Out-patients, London Hospital.
- M. H. JUPE**, B.A., M.R.C.S., L.R.C.P., F.F.R., D.M.R.E.,  
Radiologist, London Hospital.
- PETER KERLEY**, M.D., F.R.C.P., F.F.R., D.M.R.E.,  
Physician to the X-ray Department, Westminster Hospital;  
Radiologist, Royal Chest Hospital.
- C. JENNINGS MARSHALL**, M.D., M.S., F.R.C.S.,  
Surgeon, Charing Cross Hospital; Surgeon, Victoria Hospital  
for Children; Examiner in Surgery, Universities of London  
and Manchester.
- RUSSELL J. REYNOLDS**, O.B.E., M.B., B.S., F.R.C.P., F.F.R., D.M.R.E.,  
Physician in charge Departments of Radiology and Electro-  
therapy, Charing Cross Hospital; Honorary Radiologist,  
National Hospital, Queen Square; late Hunterian Professor,  
Royal College of Surgeons.

- R E ROBERTS BSc MD DPH FFR, DMRE**  
 Honorary Radiologist Liverpool Royal Infirmary Victoria  
 Central Hospital Liverpool Maternity Hospital and Liverpool  
 Radium Institute Consulting Radiologist Liverpool Heart  
 Hospital Lecturer in Radiology and in Applied Anatomy  
 (Radiological) University of Liverpool
- S COCHRANE SHANKS MD FRCP FFR**  
 Honorary Director X-ray Diagnostic Department University  
 College Hospital
- CECIL G TEALL MB ChB FR**  
 Honorary Radiologist General Hospital and Children's Hospital  
 Birmingham
- F FANE TIERNEY MRCS LRCP DMRE**  
 Assistant Radiologist Charing Cross Hospital
- I W TWINING MRCS MRCP FFR DMRE**  
 Radiologist Royal Infirmary Manchester Radiologist Christie  
 Hospital late Hunterian Professor Royal College of Surgeons  
 Lecturer in Radiology, University of Manchester
- F ROHAN WILLIAMS MD FRCP FFR DMRE**  
 Assistant Director Radiological Department St Mary's  
 Hospital Honorary Radiologist Willesden General Hospital
- J ST GEORGE WILSON MC MB ChM FRCS LRCP FRCOG**  
 Obstetric and Gynaecological Surgeon Liverpool Royal Infirmary  
 Consulting Obstetrician Walton Hospital Liverpool
- H M WORTH MRCS LRCP LDS FFR DMRE**  
 Assistant Radiologist Guy's Hospital Radiologist Guy's  
 Hospital Dental School

## PREFACE

THE OBJECT of the Editors in presenting this text book is to provide within reasonable limits a comprehensive survey of the present position of X ray diagnosis. Diagnostic radiology is becoming an increasingly complex specialty and it is difficult for one person to be equally expert in all its branches. The editors are fortunate therefore in having the help of collaborators both radiological and clinical who are distinguished in particular branches of the subject. It is hoped that this has made the work the more authoritative and that it will be of value not only to the post graduate student of radiology but also to the clinician. In conformity with this design only essential details of technique are included and the subject of X ray physics is not dealt with.

For convenience of reference the work is published in three volumes each containing as far as possible subjects of allied interest. Thus Vol I deals mainly with the thorax Vol II with the abdomen and Vol III with the skeleton and nervous systems.

The production of this the third volume has been a lengthier process than in the case of the first two largely because of the time involved in correlating the work of sixteen contributors. Here and there may be noted some overlapping between the various sections. The Editors have not regarded this as a fault if anything the reverse.

It is not possible even within the generous limits allowed by the publishers to illustrate every condition demonstrable by radiology but the illustrations chosen are it is hoped representative and give due emphasis to the common lesions met with in radiological practice. Considerable interchange of material for illustrations has taken place between the various contributors to the book and the Editors are greatly indebted for the loan of illustrations from other colleagues detailed acknowledgments of which will be found at the beginning of each volume. They are also grateful to Mr Boutall of Messrs Vaus & Crompton for the care and attention he has given to the preparation of the blocks and to Messrs Hazell Watson & Viney for their careful work with the printing.

Finally, the Editors desire to express their sincere thanks to the publishers and in particular Mr H L Jackson and Mr T Boothby for their co-operation and advice without which this book could not have come into being.

October 1939

## ACKNOWLEDGMENTS

THE EDITORS desire to make grateful acknowledgment to the following for illustrations kindly lent

Mr STANLEY BRETNALL (Fig 542) Dr G B BUSH (Fig 470) CURATOR OF ROY COLL SURG MUSEUM (Fig 381) Drs CUSHING and BAILEY (Fig 152) DR COURTNEY GAGE (Fig 545) Dr CAI HOLTEA (Fig 473) Dr F LASHOLM (Fig 73) Dr MATHER (Fig 543) Dr CRAIG MOONEY (Figs 236 238) Prof W D NEWCOMB (Figs 380 382) Mr K I NISSEN (Figs 428 435) Dr R A C RIGBY (Fig 64) Dr P ROSS (Fig 325) Dr BERTRAM SHIRTS (Figs 361 362 363) Prof SNAPPER (Fig 458) Drs A DE VET and B G ZIEFDES DES PLANTES (Fig 158) Mr PHILIP WILES (Fig 373) Dr J W WINCHESTER (Figs 364a 364b 420)

## SUMMARY OF CONTENTS

### VOL I

- CARDIOVASCULAR SYSTEM PETER KERLEY MD  
FRCP FFR DMRE  
RESPIRATORY SYSTEM E W TWining, MRCS,  
MRCP FFR, DMRE  
URINARY AND MALE GENITAL TRACTS C JENNINGS  
MARSHALL MD, MS FRCS and S COCHRANE SHANKS  
MD FRCP, FFR

### VOL II

- ALIMENTARY TRACT S COCHRANE SHANKS  
BILIARY TRACT PETER KERLEY  
ABDOMEN S COCHRANE SHANKS  
FEMALE GENITAL TRACT R E ROBERTS BSc MD  
DPH FFR DMRE and J ST GEORGE WILSON  
MC MB ChM FRCS LRCP FRCOG  
OBSTETRICS R E ROBERTS

### VOL III

- CENTRAL NERVOUS SYSTEM HUGH CAIRNS MA, DM  
BS FRCS and M H JULIF BA MRCS, LRCP  
FFR DMRE  
ACCESSORY NASAL SINUSES LABYRINTH AND  
MASTOID PROCESSES H K GRAHAM HODGSON  
CVO MB, BS FRCP FFR DMRE  
BONES AND JOINTS AND SOFT TISSUES S L BAKER  
PhD MSc MRCS, LRCP DPH, T CAMPBELL  
GOLDING, MB, ChM, MRCP DMRE, E DUFF GRAY,  
MA MD FFR, DMRE, H K GRAHAM HODGSON,  
DONALD HUNTER, MD FRCP, M H JULIF, PETER  
KERLEY, R E ROBERTS, S COCHRANE SHANKS, CYCIL G  
TEALL MB ChB, FFR, T FANE TIFENEY, MRCS,  
LRCP, DMRE, E W TWining, E ROHAN WILLIAMS,  
MD FRCP, FFR, DMRE  
TEETH AND JAWS H M WORTH, MRCS, LRCP,  
LDS, FFR, DMRE  
THE EYE S COCHRANE SHANKS and E W TWining  
CINERADIOGRAPHY RUSSELL J REYNOLDS, CBE, MB  
BS FRCP, FFR, DMRE

# CONTENTS OF VOLUME THREE

	PAGE
LIST OF CONTRIBUTORS . . . . .	v-vi
PREFACE . . . . .	vii
SUMMARY OF CONTENTS . . . . .	ix

## PART ONE

### CENTRAL NERVOUS SYSTEM

CHAPTER

I. GENERAL TECHNIQUE AND PATHOLOGY . . . . .	3
II. THE RADIOLOGICAL FEATURES OF THE CRANIUM, NORMAL AND PATHOLOGICAL . . . . .	30
III. VENTRICULOGRAPHY AND ENCEPHALOGRAPHY . . . . .	71
IV. INTRACRANIAL LESIONS . . . . .	126
V. ANOMALIES AND DISEASES OF THE SKULL . . . . .	165
VI. THE SPINAL CORD . . . . .	185

## PART TWO

### THE ACCESSORY NASAL SINUSES, LABYRINTH, AND MASTOID PROCESS

VII. ANATOMY OF THE ACCESSORY NASAL SINUSES . . . . .	201
VIII. TECHNIQUE OF EXAMINATION . . . . .	207
IX. DISEASES OF THE ACCESSORY NASAL SINUSES . . . . .	217
X. THE USE OF IODISED OIL IN THE DIAGNOSIS OF NASAL SINUS DISEASE . . . . .	226
XI. ANATOMY OF THE TEMPORAL BONE . . . . .	230
XII. THE LABYRINTH AND PETROUS BONE . . . . .	234
XIII. THE MASTOID PROCESS . . . . .	237

## PART THREE

### THE BONES AND JOINTS AND SOFT TISSUES

#### SECTION I

#### THE NORMAL BONES AND JOINTS OF THE EXTREMITIES

XIV. NORMAL BONES AND JOINTS . . . . .	251
XV. NORMAL BONES AND JOINTS OF THE UPPER EXTREMITY . . . . .	255
XVI. NORMAL BONES AND JOINTS OF THE LOWER EXTREMITY . . . . .	271

## THE NORMAL SPINE AND PELVIS

CHAPTER

## XVII THE NORMAL VERTEBRAL COLUMN AND PELVIS

PAGE

293

## SECTION II

## THE GENERAL PATHOLOGY OF BONE

XVIII	THE GENERAL PATHOLOGY OF BONE	307
XIX	THE GENERAL PATHOLOGY OF BONE ( <i>continued</i> )	324

## SECTION III

## CONGENITAL DEFORMITIES OF BONES AND JOINTS

XX	GENERAL CONSIDERATIONS	341
XXI	CONGENITAL MALFORMATIONS OF THE SKULL	343
XXII	CONGENITAL MALFORMATIONS OF THE SPINE AND PELVIS	350
XXIII	CONGENITAL MALFORMATIONS OF THE UPPER AND LOWER LIMBS	356

## SECTION IV

## TRAUMATIC LESIONS OF BONES AND JOINTS

## FRACTURES OF THE FACIAL BONES

XXIV	FRACTURES AND DISLOCATIONS	36
XXV	LATE SEQUELÆ OF TRAUMA OF BONES AND JOINTS	389
XXVI	FRACTURES OF THE FACIAL BONES	396

## SECTION V

## INFLAMMATORY DISEASES OF BONES AND JOINTS

XXVII	PERIOSTITIS AND OSTFOMELITIS	401
XXVIII	TUBERCULOSIS OF BONE	414
XXIX	TUBERCULOSIS OF JOINTS	423
XXX	SYPHILIS OF BONES AND JOINTS	436
XXXI	UNCOMMON INFLAMMATORY DISEASES OF BONE	450
XXXII	ACUTE AND CHRONIC ARTHRITIS	456

## SECTION VI

## OSTEOCHONDRITIS

XXXIII	OSTEOCHONDRITIS	474
--------	-----------------	-----

## SECTION VII

STATIC AND PARALYTIC LESIONS—THE INTERVERTEBRAL DISCS—ORTHOPEDIC  
OPERATIONS

CHAPTER		PAGE
XXXXV	STATIC AND PARALYTIC LESIONS OF BONES AND JOINTS	493
XXXXVI	THE INTERVERTEBRAL DISCS	495
XXXXVII	ORTHOPEDIC OPERATIONS	504

## SECTION VIII

## DISEASES OF BONES AND JOINTS OF CONSTITUTIONAL OR UNKNOWN ORIGIN

XXXXVIII	DISEASES DUE TO DISTURBANCE OF GROWTH	513
XXXXIX	DISEASES DUE TO DISTURBANCES OF NUTRITION	522
XXXXX	DISEASES DUE TO DISTURBANCE OF METABOLISM	535
LI	DISEASES DUE TO DISTURBANCE OF THE ENDOCRINE SYSTEM	542
LI	BONE AND JOINT CHANGES IN IONIC CONDITIONS	556
LI	BLOOD DISEASES WITH ASSOCIATED BONE CHANGES	559
LIII	CARCINOMATOSIS AND MULTIPLE MYELOMATOSIS OF BONES	565
LIV	DISEASES OF UNKNOWN ORIGIN	568

## SECTION IX

## TUMOURS AND CYSTS OF BONE

XLV	SIMPLE TUMOURS OF BONE	587
XLVI	MALIGNANT TUMOURS OF BONE	605
XLVII	CYSTS OF BONE	636

## SECTION X

## THE SOFT TISSUES

XLVIII	CALCIFICATION AND OSSIFICATION IN THE SOFT TISSUES	644
XLIX	OTHER LESIONS OF THE SOFT TISSUES	660

## SECTION XI

## LOCALISATION OF FOREIGN BODIES

L	THE LOCALISATION OF FOREIGN BODIES	672
---	------------------------------------	-----



CHAPTER	THE NORMAL SPINE AND PELVIS	PAGE
XVII	THE NORMAL VERTEBRAL COLUMN AND PELVIS	293

## SECTION II

## THE GENERAL PATHOLOGY OF BONE

XVIII	THE GENERAL PATHOLOGY OF BONE	302
XIX	THE GENERAL PATHOLOGY OF BONE ( <i>continued</i> )	324

## SECTION III

## CONGENITAL DEFORMITIES OF BONES AND JOINTS

XX	GENERAL CONSIDERATIONS	341
XXI	CONGENITAL MALFORMATIONS OF THE SKULL	343
XXII	CONGENITAL MALFORMATIONS OF THE SPINE AND PELVIS	350
XXIII	CONGENITAL MALFORMATIONS OF THE UPPER AND LOWER LIMBS	356

## SECTION IV

## TRAUMATIC LESIONS OF BONES AND JOINTS

## FRACTURES OF THE FACIAL BONES

XXIV	FRACTURES AND DISLOCATIONS	367
XXV	LATE SEQUELÆ OF TRAUMA OF BONES AND JOINTS	389
XXVI	FRACTURES OF THE FACIAL BONES	396

## SECTION V

## INFLAMMATORY DISEASES OF BONES AND JOINTS

XXVII	PERIOSTITIS AND OSTEOMYELITIS	401
XXVIII	TUBERCULOSIS OF BONE	414
XXIX	TUBERCULOSIS OF JOINTS	423
XXX	SYPHILIS OF BONES AND JOINTS	436
XXXI	UNCOMMON INFLAMMATORY DISEASES OF BONE	450
XXXII	ACUTE AND CHRONIC ARTHRITIS	456

## SECTION VI

## OSTEOCHONDRITIS

XXXIII	OSTEOCHONDRITIS	474
--------	-----------------	-----

## SECTION VII

STATIC AND PARALYTIC LESIONS—THE INTERVERTEBRAL DISCS—ORTHOPEDIC  
OPERATIONS

CHAPTER		PAGE
XXXIV	STATIC AND PARALYTIC LESIONS OF BONES AND JOINTS	493
XXXV	THE INTERVERTEBRAL DISCS	498
XXXVI	ORTHOPEDIC OPERATIONS	504

## SECTION VIII

## DISEASES OF BONES AND JOINTS OF CONSTITUTIONAL OR UNKNOWN ORIGIN

XXXVII	DISEASES DUE TO DISTURBANCE OF GROWTH	513
XXXVIII	DISEASES DUE TO DISTURBANCES OF NUTRITION	522
XXXIX	DISEASES DUE TO DISTURBANCE OF METABOLISM	535
XL	DISEASES DUE TO DISTURBANCE OF THE ENDOCRINE SYSTEM	542
XLI	BONE AND JOINT CHANGES IN IONIC CONDITIONS	556
XLII	BLOOD DISEASES WITH ASSOCIATED BONE CHANGES	559
XLIII	CARCINOMATOSIS AND MYELITIC MYELOMATOSIS OF BONES	565
XLIV	DISEASES OF UNKNOWN ORIGIN	568

## SECTION IX

## TUMOURS AND CYSTS OF BONE

XLV	SIMPLE TUMOURS OF BONE	587
XLVI	MALIGNANT TUMOURS OF BONE	605
XLVII	CYSTS OF BONE	636

## SECTION X

## THE SOFT TISSUES

XLVIII	CALCIFICATION AND OSSIFICATION IN THE SOFT TISSUES	644
XLIX	OTHER LESIONS OF THE SOFT TISSUES	662

## SECTION XI

## LOCALISATION OF FOREIGN BODIES

L	THE LOCALISATION OF FOREIGN BODIES	6
---	------------------------------------	---

*PART FOUR*

## THE TEETH AND JAWS

## CHAPTER

- LI THE NORMAL TEETH AND JAWS
- LII UNERUPTED AND SUPERNUMERARY TEETH
- LIII INFLAMMATORY DISEASES OF THE PERIODONTAL MEMBRANE  
AND ALVEOLUS
- LIV INJURIES TO THE TEETH AND JAWS
- LV CYSTS AND TUMOURS OF THE TEETH AND JAWS
- LVI INFLAMMATORY AND OTHER DISEASES OF THE JAWS
- LVII DISEASES OF THE TEMPORO-MANDIBULAR JOINT AND  
ANTRUM

*PART FIVE*

## THE EYE

- LVIII THE EYE

*PART SIX*

## CINERADIOGRAPHY

- LIX INTRODUCTION
- LX METHODS OF CINERADIOGRAPHY
- LXI CLINICAL APPLICATIONS OF CINERADIOGRAPHY
- INDEX

VOLUME III

*PART ONE*

CENTRAL NERVOUS SYSTEM

BY

HUGH CAIRNS, M A , D M , B S , F R C S , AND M H JUPE  
M R C S , L R C P F F R , D M R E

# A TEXT-BOOK OF X-RAY DIAGNOSIS

## PART ONE

### CENTRAL NERVOUS SYSTEM

#### CHAPTER I

#### GENERAL TECHNIQUE AND PATHOLOGY

RADIOGRAPHIC EXAMINATION of the skull and its contents is now securely established as a most important method of investigating all cerebral disorders associated with a rise of intracranial pressure, or with symptoms indicating a progressive course. Almost every form of expanding lesion within the cranium whether tumour, large aneurysm or infantile hydrocephalus, will produce in time local or diffuse changes in the radiographic appearance of the cranium and the evidence thus furnished sometimes provides a peculiarly direct indication of the presence and situation of the lesion and even of its nature.

The localisation of expanding intracranial lesions, such as tumour, depends primarily on a careful study of the patient's symptoms and an accurate and detailed neurological examination. However, the mind of the patient may be so disturbed by cerebral damage that investigation of the functions of many of his nervous pathways becomes almost impossible, in other cases the tumour may develop in a "silent" area of the brain and give no localising signs until its advancing margins perhaps far from the origin of the lesion begin to disturb known pathways, or the tumour may so distort the brain as to produce damage in distant parts of it, with the result that the patient has physical signs susceptible of faulty interpretation as to the situation of the lesion—as for example, ipsilateral hemiparesis in a tumour of one cerebral hemisphere. There are, indeed, so many pitfalls that even the most expert neurologist will admit his inability to furnish in every case an accurate clinical diagnosis.

To this problem of localisation of intracranial tumours radiology has contributed much.<sup>1</sup> It brings evidence of an entirely different kind from that

<sup>1</sup> In 1931 from plain X-ray studies Sosman was able to predict the location of the intracranial tumour in 50 per cent of the cases submitted from Cushing's clinic and to give an accurate opinion as to the histological type of the tumour in 75 per cent. In 4 per cent of the same series radiograms suggested the presence of tumour where none was found.

*Bibliography.* A bibliography is given at the end of Chapters II to VI. It is not comprehensive but is intended as a guide to more detailed reading.

obtained by clinical examination evidence of a direct visual order which provides a cross reference on the map of topographical diagnosis. The value of ventriculography and of its substitute ventricular estimation, will be referred to later. In this chapter we will deal with simple X ray examination. Fig 1 shows a large left fronto temporal meningioma in a patient whose predominant clinical signs when she first came under expert neurological care were papilloedema left facial palsy of peripheral type and bilateral



FIG 1—O s y ng left fronto temporal mening oma w thout l yperostosis Case No 66

pyramidal signs. The first diagnosis was tumour of the pons. Radiograms were then taken and the shadow of the tumour was seen. Shortly after this the facial palsy almost disappeared and further consideration of the history of illness left little doubt that the lesion shown by radiography was responsible for the patient's symptoms. The clinical problems presented by this case were not insoluble but without the help of radiograms of the skull a false conclusion might have been reached that operation gave no chance of relief or operation might have been directed towards the wrong part of the brain.

We have had other cases of this type in which the first clue to accurate localisation of the tumour has been from the radiograms. There are too

cases in which the first definite evidence that the patient is suffering from an intracranial tumour and not from some other nervous disorder has been provided by radiographic examination. Sometimes as in the case described above radiograms indicate not only the situation but also the nature of the tumour. It must not be concluded, however, that radiography can often help to this extent. Direct evidence of the situation of the tumour is provided by simple radiograms in no more than 10 to 15 per cent of cases and then the evidence may be of such an indefinite nature that it can be considered of importance only when taken in conjunction with the clinical signs and should



FIG. 2.—Calcified plaque of the cerebral

be rejected if not supported by them. A doubtful fleck of calcification seen in a radiogram becomes significant when the clinical picture is in accord with its situation and when further specially taken radiograms may confirm its presence. Such a radiographic finding may not infrequently suggest to the surgeon that the tumour arises in an area 1 or even 2 inches in front of or behind the area in which it was thought to be and the osteoplastic exploration is accordingly shifted forwards or backwards from the site originally planned.

Even when it is nebulous radiological evidence in a difficult case will often start the observer on a new line of thought as a result of which the significance of the previously observed clinical signs is gauged more surely and the correct diagnosis reached. Radiologists can greatly improve their interpretation of

radiograms of the head by acquiring a working knowledge of the symptomatology and pathology of intracranial tumours

These principles are of particular importance at the present time, when the perfection of technique and increasing popularity of radiological examination are revealing more and more novel appearances on and inside the skull. There is a strong temptation to regard appearances that are novel as abnormal, and thus as the cause of the patient's symptoms. When calcification of the pineal gland was first observed radiologically it was hailed by some as evidence of insanity for it so happened that the skulls in which this calcification was observed belonged to the inmates of an asylum. Even within the past year we have seen a patient with retrobulbar neuritis who was referred on account of a calcified mass within the cranium and this mass marked with an arrow in the radiograms the patient brought with him, was the shadow of a normal pineal gland. The bony processes that sometimes join the anterior to the posterior clinoid processes, and which in radiograms appear to form a bridge above the pituitary gland have been claimed as the cause of epilepsy, of idiocy, of diabetes insipidus, and of other maladies but further experience has resulted in the conclusion that the bridge is more apparent than real, since it does not extend into the diaphragma sellæ and that it is within the limits of normal. More recently it has been claimed that calcified plaques seen radiologically in the falx cerebri are the cause of a syndrome of which the major symptom is headache. Fig 2 shows a large calcified plaque of the falx in a patient who had suffered from headaches for months. These headaches subsided after the removal of a large right parietal glioma.

We have stressed this point because in recent years there have been more negative operative explorations for intracranial tumour resulting from faulty radiological diagnosis than from any other cause. The following examples of such negative explorations have come within our knowledge.

(1) A man with progressive mental symptoms was found to have a calcareous plaque on the falx cerebri (similar to the one in Fig 2) and was explored with a pre operative diagnosis of meningioma of the falx.

(2) In a patient suffering from headaches slight left hemiparesis and hemianalgesia radiograms showed in the right parietal bone a circular area of diminished density, and behind this area numerous large diploic channels. From this evidence it was thought at neurological consultation that the patient probably had a meningioma. A right lateral osteoplastic exploration was done. No tumour was found, but only small convolutions covered by large subarachnoid spaces. Increasing experience has shown that the appearances seen in the radiograms in this and the preceding case are really within normal limits.

(3) A patient suffering from progressive mental disturbance and fits was found to have a large area of resorption in the left frontal region, and a radiological diagnosis of left frontal meningioma was made. At operation it was



found that the resorption was due to an unusually large congeries of Pacchionian granulations. The symptoms were due to severe chronic leptomenigitis, probably the result of chronic alcoholism (fig. 3).

There is clearly need for wider knowledge of the normal radiographic appearances of the head.

At this stage it is important to stress the fallacy of what might be called haphazard radiography of the skull. For example a patient with progressive



FIG. 3.—Extensive resorption of internal table of frontal bone associated with numerous Pacchionian granulations in a case of chronic leptomenigitis. Case No. 2008.

right hemiparesis is said to show no radiological evidence of intracranial disease when the lateral pictures have been taken with the right side of the head against the film although the probabilities are that the lesion is in the left cerebral hemisphere. Examples of this type could be multiplied from our experience. The responsibility is borne by the clinician but the radiologist, if he wishes to get the best results will also play his part in seeing that the type of radiographic examination is governed by the presenting clinical signs.

### TECHNIQUE

We do not propose to give a detailed description of apparatus or technique. Each radiologist will know best how to use his own plant and there is at

present such lack of standardisation that what serves for one plant in the matter of technique, measurement, and adjustment of apparatus will not necessarily be effective for another. Good work of a routine character can be done with any standard radiographic couch and fine-focus tube, but for certain

special projections apparatus of the type designed by Lysholm is essential (Fig 4). In this apparatus the Bucky diaphragm can be moved in any plane, the tube, travelling on a pivoting overhead arc, is always centred the tube table distance is constant and the angles of all movements can be measured.

If the radiation is too hard, the rays may pass through tumours which are scantily calcified. The radiation should be as soft as possible without prolonging the exposure and risk to the patient's skin from rays that are too soft to penetrate to the film. Skull examinations entail many films and after simple radiograms have been taken further exposures are often necessary for encephalography and ventriculography. For this reason, a filter should always be used and we have found 1 mm of aluminium satisfactory. The variation of skulls in thickness and density cannot yet be predicted by clinical means the first film should therefore be examined before the others are taken.

Most examinations are made with the patient lying down, as he is more likely to keep still in this position than if sitting up, especially if he is weak, stuporous, or disturbed in his balance. In "direct sella"

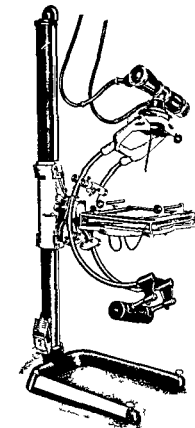


FIG 4 — The Lysholm table

films the sitting position is frequently used, but this examination can also be done with the patient lying down.

Some form of centring device is essential, and the smallest possible diaphragm should be employed. We do not fix the head unless the patient is restless, when a strap is used in preference to clamps.

The value of stereoscopy cannot be overstressed. A few highly expert radiologists work with single films, but most employ stereoscopy for almost every projection. Only by this method is it possible to learn the anatomical significance of the many lines and shadows at the base of the skull.

With stereoscopic vision the side or part of the skull nearest to the film is seen in detail, uncomplicated by the shadows of the opposite side, and thus small alterations in density and pattern can be easily distinguished. In some projections it is better to carry out stereoscopy in a longitudinal direction, while in others it should be transverse; the films should always be viewed both orthoscopically and pseudoscopically.

Many projections have been described. Some of them have little practical value, and we propose to describe only the more common, those that are used for routine examinations, and those for certain special regions of the skull. It is scarcely necessary to mention that individual cases may require a departure from the standard projections, as in the case of a hyperostosis, which may be adequately displayed only by tangential study of the inner table of the skull.

### ROUTINE EXAMINATION

In the routine examination of patients with intracranial lesions lateral, postero-anterior, and antero-posterior projections are made. In describing the

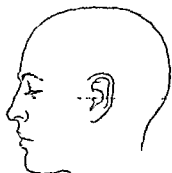


FIG. 5.—Orbito-meatal line.

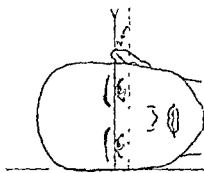


FIG. 6.—Lateral projection. The dotted line represents the orbito-meatal line.

various positions, the tilt of the tube is measured by the angle the central ray forms with the orbito-meatal line. This line (also referred to as the "eye-ear" line, the Frankfurter horizontal plane, or Reid's baseline) joins the upper edge of the external auditory meatus with the lowest point of the inferior orbital ridge (Fig. 5).

**Lateral Projection.**—Both right and left lateral stereoscopic films should be taken, otherwise important radiographic signs may be missed. In meningiomas of the convexity, for example, the finer changes in the bone can only be seen when the corresponding side of the head is against the film. Not infrequently the clinician is unable to tell the radiologist with any degree of certainty on which side of the head the lesion lies.

The patient is placed prone with the side of the head to be examined

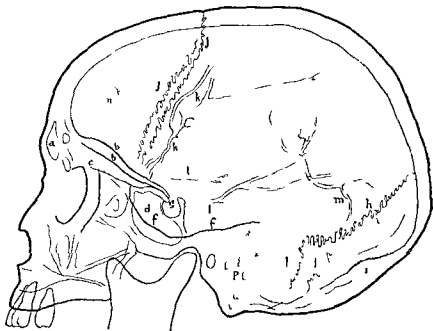


FIG 7.—Lateral projection of skull. Normal appearance. (a) Frontal sinus (b) Roof of each orbit (c) Cribriform plate of ethmoid (d) Sphenoidal air sinus (e) Floor of middle fossa (f) Sella turcica (g) Lambdoid suture (h) Internal occipital protuberance (i) Coronal suture (j) Meningeal groove (anterior branch) (k) Meningeal groove (posterior branch) (l) Meningeal groove of opposite side of skull (m) Dural vessels (n) Mastoid air cells

towards the film. The sagittal axis of the skull is parallel to the surface of the table. The central ray is adjusted over a point 2 cm. above the orbito-meatal line and 2 cm. in front of the external auditory meatus (Fig. 6). This point is immediately over the sella turcica. It is preferable that the stereoscopic shift should be in the vertical direction.

Thus projection (Fig. 7) displays the shape, thickness, and texture of the cranial vault, the sutures, the meningeal and diploic channels, the sella turcica, areas of calcification in the pineal body, choroid plexuses, or elsewhere. The base of the skull is seen in profile.

**Postero-anterior Projection.**<sup>1</sup>—The patient is face downwards with his nose and forehead on the surface of the table. The external auditory meatuses are equidistant from the film. The central ray is directed to pass through the nasion, but is not absolutely vertical, the tube being tilted 20 degrees towards the vertex (Fig. 8). Stereoscopy should be in a transverse direction.

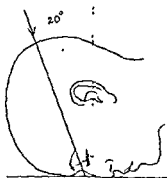


FIG. 8.—Postero anterior projection.

In a satisfactory picture (Fig. 9) the crests of the petrous bones should lie at the lower margins of the orbits, or slightly below this, so as not to disturb the rest of the picture. A good view is thus obtained of the frontal bone, the anterior fossa, the orbits, the lesser wings of the sphenoid, and the accessory sinuses of the nose. The anatomical interpretation of lines seen in the orbit is shown in Fig. 56 (see p 56). The foramen rotundum can often be seen; the optic foramen rarely.

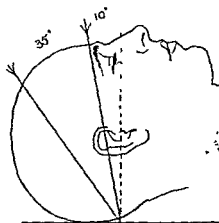


FIG. 10.—Antero posterior projections (see text).

**Antero-posterior Projections.**<sup>2</sup>—It is our custom to dispense with stereoscopy and to take two single films. The patient lies on his back with the occiput towards the film and the external auditory meatuses equidistant from the film. The chin is depressed slightly towards the sternum so that the orbito-meatal line is vertical. In all cases the central ray should pass through the occipital protuberance. The radiogram can then be taken without a tilt or with various degrees of tilt towards

<sup>1</sup> The standard terminology, now almost universally adopted, is to mention last that part of the head which is against the film. Thus "postero-anterior" indicates that the brow is against the film; "antero-posterior" indicates that the occipital region is against the film.

<sup>2</sup> This projection with varying degrees of tilt has been suggested by many different authors (Grashey, Towne, Lysholm, and others).

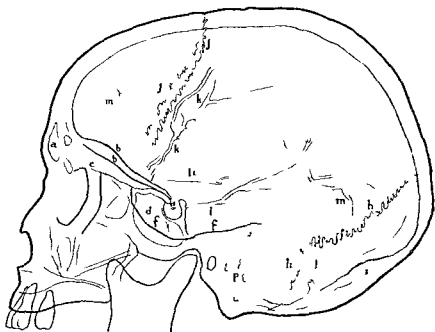


FIG 7—Lateral projection of skull Normal appearance (a) Frontal sinus (b b) Roof of each orbit (c) Cribriform plate of ethmoid (d) Sphenoidal air sinus (f f) Floor of middle fossa (g) Sella turcica (h) Lambdoid suture (i) Internal occipital protuberance (j) Coronal suture (k) Meningeal groove (anterior branch) (l) Meningeal groove (posterior branch) (m) Meningeal groove of opposite side of skull (n) Diploic vessels (p) Mastoid air-cells

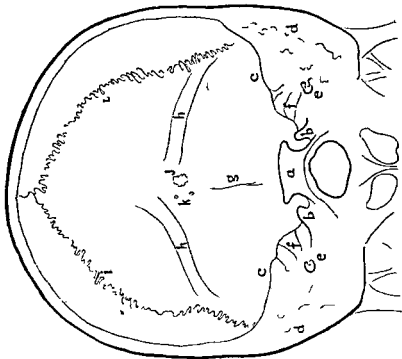
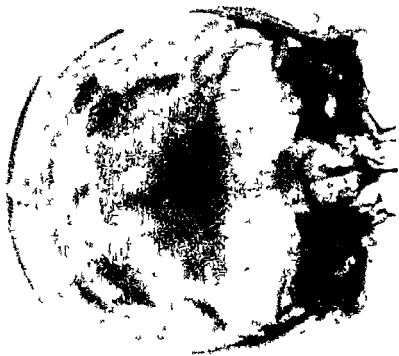


Fig 11 — Antero posterior projection (25 degrees tilt)  
 (b) Anterior clinoid process (c) Crest of petrous bone  
 occipital crest (f) Transverse sinus (g) Lamboid suture

Normal appearance (a) Dorsum sellae and posterior clinoid processes  
 (d) Mastoid cells (e) Cochlea (f) Internal auditory canal (g) Internal  
 (j) Funeal body (k) Occipital emissary foramina

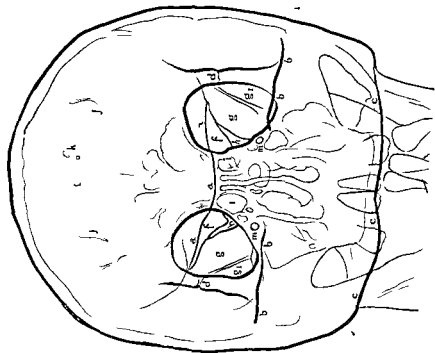


FIG 9.—Postero anterior projection of skull. Normal appearance (a) Floor of anterior fossa (b) Crest of petrous bone (c) Floor of posterior fossa (d) Fronto-malar suture (e) Lesser wing of sphenoid continued medially as anterior clinoid process (f) Greater wing of sphenoid (g) Frontal wing of sphenoid (h) Sphenoidal fissure (i) Ethmoidal air cells (j) Frontal diploic vessels (k) Pacchionian depressions (m) Foramen rotundum (n) Foramen ovale (o) Foramen spinosum (p) Foramen transversum (q) Foramen lacerum (r) Foramen magnum (s) Foramen condyloideum (t) Foramen condyloideum (u) Foramen condyloideum (v) Foramen condyloideum (w) Foramen condyloideum (x) Foramen condyloideum (y) Foramen condyloideum (z) Foramen condyloideum



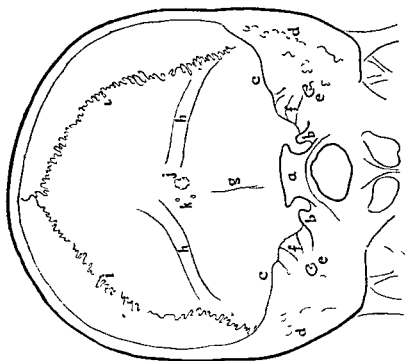
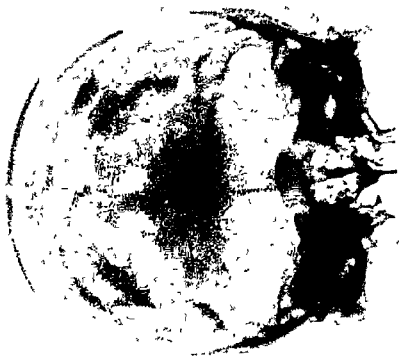


FIG. 11.—Antero posterior projection (25 degrees tilt). Normal appearance. (a) Dorsum sellae and posterior clinoid processes. (b) Anterior clinoid process. (c) Crest of petrous bone. (d) Mastoid cells. (e) Cochlea. (f) Internal auditory canal. (g) Internal occipital crest. (h) Transverso sinus. (i) Lambdoid suture. (j) Pincal body. (k) Occipital emissary foramina.



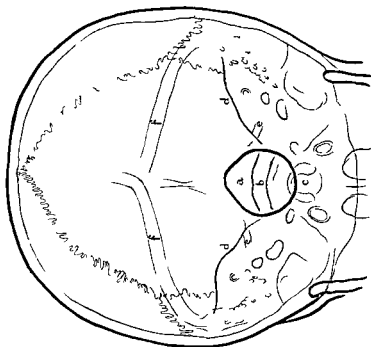
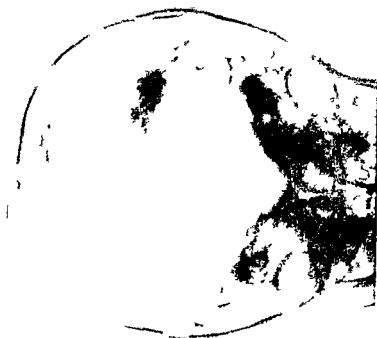
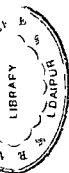


FIG 1<sup>a</sup>—Frontal process of atlas (a) Occipital process (b) Posterior arch of atlas (c) Occipital process (d) Crest of petrous bone (e) Internal auditory canal (f) Transverse sinus

the head. We have found that most information is obtained by taking one film with a tilt of 25 degrees towards the head, and a second film with a tilt of 35 degrees in the same direction (Fig. 10). The first of these projections is designated "antero-posterior" (Fig. 11), the second "fronto-occipital" (half-axial antero-posterior) (Fig. 12). These views show the interior of the posterior part of the skull, the pineal shadow, the outline of the lower part of the tabular part of the occipital bone, and the occipital emissary canals; the anterior and posterior clinoid processes are seen in one or both views, the shadows of the petrous bones are seen above the orbits. In the fronto-occipital picture the foramen magnum is well seen. In many instances important confirmatory evidence of radiological signs observed in other views may be obtained. In cases where in the lateral projections pressure deformity of one anterior clinoid process is suspected, these projections will supply important confirmatory evidence, especially of lateralisation.

### SPECIAL EXAMINATIONS<sup>1</sup>

**Occipito-frontal Projection (half-axial postero-anterior projection).**—If there is difficulty in obtaining the fronto-occipital projection on account of shortness of the patient's neck, then similar information may be obtained from a projection in the reverse direction.

**Submento-vertical Projection (axial projection).**—The patient lies on his back. The highest point of the vertex rests on the top of the Bucky diaphragm, and the central ray is directed through the submental region at the hyoid bone (Fig. 13). The external auditory meatuses should be equidistant from the film. The central ray should make an angle of 90 degrees with the orbito-meatal line, and, if the patient cannot extend the head sufficiently, the tube must be tilted until the correct angle is obtained.

This projection is not difficult to procure, even in fat patients with short necks. To get the head extended far enough, it is necessary to have the shoulders higher than the Bucky diaphragm, and it will help materially if the buttocks are raised higher than the shoulders. It is advisable to take stereoscopic films; the traverse of the tube should be transverse.

This picture shows the foramina magnum, spinosum and ovale, the petrous bone with the ossicles of the ear, the meningeal grooves in their proximal parts, and other details at the base of the skull (Fig. 14).

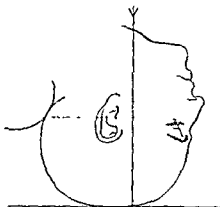


FIG. 13.—Submento vertical axial projection

<sup>1</sup> For a fuller account of special projections, the reader is referred to the works of Schuller, Lysholm, and Sante.



Fig. 14—Sphenoid, vertical projection. Normal appearance. (a) Ioramen magnum (b) Olfactory process (c) Occipital condyles (d) Jugular foramen (e) External auditory meatus (f) Middle ear with ossicle (g) Internal auditory canal (h) Cochlea (i) Semicircular canals (j) Carotid canal (k) Foramen spinosum and proximal part of middle meningeal groove (l) Foramen ovale (m) Anterior wall of middle fossa (n) Lateral wall of maxillary antrum (o) Lateral wall of orbit (p) Maxillary antrum



This view shows the optic foramen through the upper medial part of the orbit and, alongside it, the anterior clinoid process. The greater and lesser wings of the sphenoid are also seen (Fig 17)

**Lateral Projection for Porus Acusticus**—The patient lies prone with his

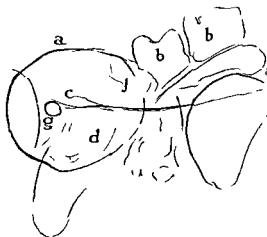
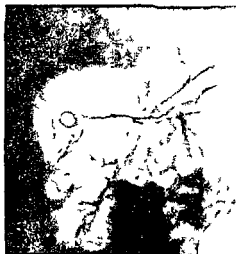


FIG 17—Projection for right optic foramen. Normal appearance. (a) Upper margin of orbit (ai) Upper margin of left orbit (b) Frontal sinuses (c) Floor of anterior fossa continuing into the lesser wing of sphenoid laterally (d) Floor of orbit (g) Optic foramen (j) Ethmoidal air cells

head in the lateral position and the side to be examined against the Bucky diaphragm. The tube is tilted 15 degrees cranially (Fig 18). The central ray passes through a point 1 inch above and 2 inches behind the external auditory meatus of the uppermost ear (Fig 19)

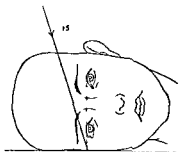


FIG 18—Lateral projection for porus acusticus

This picture, used by *Schuller, Henschen* and others for the radiographic examination of cerebello pontine angle tumours, shows the shadow of the internal auditory meatus superimposed on that of the external meatus. It is doubtful whether this projection and the one that follows are as useful for showing the internal meatus as the fronto-occipital projection already described.

**Vertical Projections for Porus Acusticus**

**and Petrous Bone**—In these projections the central ray is perpendicular to the long axis of the petrous bone. *Steniers* method is to place the patient prone, the head is rotated so that the sagittal plane of the skull is at an

angle of 45 degrees to the film. The orbito mental line is perpendicular to the film. The central ray is directed to the external occipital protuberance and the tube is moved caudally 12 degrees (Fig 20). Other methods of investigating the petrous bone at right angles to its long axis have been described by *Mayer*, *Schuller Carr* and others.

**Projections for Upper Half of the Orbits**—The patient is prone with the chin resting on the couch and the forehead raised so that the orbito mental line makes an angle of 35 degrees with the vertical.

Usually this means that the weight is taken on the chin and the tip of the nose just touches the surface of the couch. The central ray is directed vertically downwards through the orbit (Fig 21). If a Lysholm table is used the nose and forehead rest on the table and the tube is tilted 35 degrees towards the vertex. Stereoscopic films should be taken with transverse shift.



FIG 19—Lateral projection for porus acusticus. Normal appearance. The outline of the internal auditory meatus is seen lying inside that of the external auditory canal.

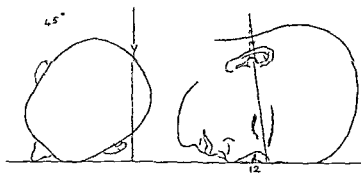


FIG 20—Vertical projection for petrous bone (Stenvers).

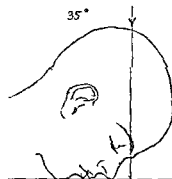


FIG 21—Projection for upper half of orbit.

By this method the upper part of the orbit is seen more completely than in the routine postero anterior view, and the accessory nasal sinuses show well (Fig 22). The view will often help in cases where sphenoidal ridge tumours are suspected.

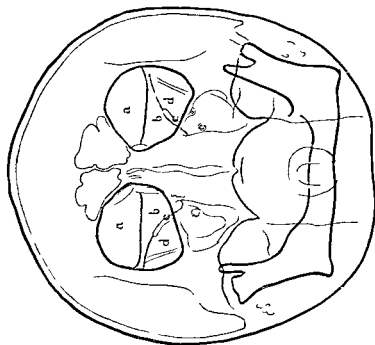


Fig. 22—1 rejection for 1st half of orbit. Normal appearance. (a) Roof of orbit. (b) Lesser wing of sclerite. (c) Greater wing of sclerite. (d) Sclerite.



Planigraphy or Tomography, introduced by *Bocage*, *Ziedses des Plantes*, and others, and greatly simplified by *E H Tuning*, is a method of radiography which aims at displaying a selected plane of the patient's body. In skull work this method can be used with advantage to show structures that are obscured in simple radiograms (Fig 158). The cribriform plate, for example, can be shown in a lateral planigram of the skull without any of the lines of the orbital roof which in simple radiograms make identification of the cribriform plate difficult.

### INTRACRANIAL PATHOLOGY IN RELATION TO RADIOLOGY

Before describing radiological appearances in disease, it is necessary to refer briefly to those aspects of intracranial pathology which bear on radiological diagnosis.

(a) *Rise of Intracranial Pressure*—With few exceptions intracranial tumours and other expanding intracranial lesions produce a rise of intracranial pressure. This may be so intense that the patient becomes severely ill before any change in the skull can be shown by X rays, but, as a general rule, when there is rise of intracranial pressure for a period of six months or more, radiographic signs appear. The rise of pressure within the cranium may be produced almost entirely by the bulk of the tumour and oedema of the surrounding brain, but more often is due to the fact that the tumour blocks the outflow of cerebro-spinal fluid from the ventricular system, and so produces hydrocephalus. This is particularly liable to occur in lesions of the posterior fossa, midbrain, and third ventricle. Both lateral ventricles become dilated and the intraventricular pressure is raised from the normal 150 mm of water, in some cases to as much as 1,000 mm. This rise of pressure is communicated to all parts of the cranium.

Its effects upon the skull vary with the age of the patient and the condition of the sutures. In infants and young children rise of pressure produces separation of the sutures and enlargement of the head (Fig 23). This is tantamount to partial decompression, and may delay the onset of symptoms, thus a child with a benign tumour and a large head may have quite a short history of illness. After the age of 15 the bones begin to join and the skull cannot expand to any great extent.<sup>1</sup> General rise of pressure then tends to produce excessive resorption of those parts of the vault that overlie the cerebral convolutions, and this in time gives rise to a characteristic "beaten silver" appearance (Fig 24). The bone is thinned over the convolutions, and the ridges of more dense bone correspond to the sulci. Similar markings appear on the base of the skull, in the middle and anterior fossæ,<sup>1</sup> but these cannot be adequately displayed in radiograms.

Generalised thinning of the skull occurs with severe and long continued

<sup>1</sup> Naturally, no markings occur over the cerebellum where there are no convolutions.



FIG 23 —Separation of sutures due to cerebellar medulloblastoma in a child aged 8 Case No. L H 31870/1936

internal hydrocephalus from any cause, but is of little value in radiological diagnosis owing to the great variations in the normal thickness of the skull. With rise of intracranial pressure the diploic channels and the foramina of the emissary veins become enlarged; this is doubtless due to the rise of intracranial venous pressure, which is a constant accompaniment of raised intracranial pressure.

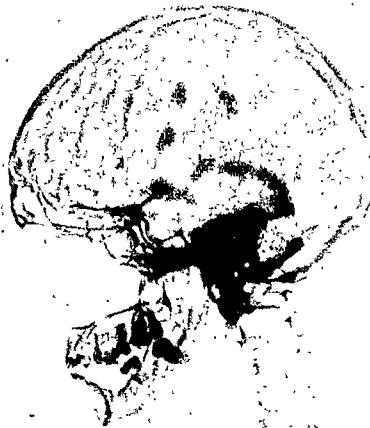


FIG. 24.—Convolutional markings in a patient with internal hydrocephalus due to adhesions in the region of the fourth ventricle. The sella turcica also is partially destroyed.  
Case No. L.H. 41781/1938.

In adults the earliest and most constant evidence of rise of intracranial pressure is found in the sella turcica. From the manner in which it projects into the cranial cavity the dorsum sellæ, with the posterior clinoid processes, is particularly vulnerable to increase of pressure. It tends to disappear and at the same time the fossa may become enlarged, even when the lesion causing the rise of intracranial pressure is remote. The mechanisms by which sellar changes are produced will be described later.

Owing largely to the dural partitions, a rise of intracranial pressure is not

equally distributed throughout all parts of the cranium. Tumours of the pituitary gland produce enlargement of the sella turcica usually without any pressure effect upon the rest of the cranium. In benign tumours of slow growth in other parts of the cranium especially in children radiological examination may reveal evidence of local increase of pressure in the form of localised projection and excessive thinning of the bone. The bone always gives way before the dura. Fig. 25 shows an example of local rise of pressure.



FIG. 25.—Bulging and thinning of the right temporal region from a right temporal astrocytoma in a child aged  $7\frac{1}{2}$ . The radiogram also shows a cyst in the left ventricle. Case No. 1371.

(b) **Displacements**—Intracranial tumours and other expanding intracranial lesions tend to produce distortion of the brain. A tumour arising in one cerebral hemisphere may displace the falx, lateral and third ventricles and even the midbrain to the opposite side (Fig. 19). The phenomenon is of great importance in its application to diagnosis by means of ventriculography, but it also has some bearing on simple radiological diagnosis on account of the displacement of calcified contents of the skull. The pineal gland is usually

calcified in adult life and its displacement in any direction may be recognised radiologically. There is often calcification in part of the choroid plexus of the lateral ventricle and if this is visible in an abnormal position it will provide indirect evidence of the site of the tumour. The falx cerebri is sometimes calcified and visible in radiograms and its deflection by a tumour of one cerebral hemisphere will then be a valuable observation.

(c) **Calcification of Tumours**—Gliomas, epidermoid cysts, meningiomas, some blood vessel tumours, tuberculomas, teratomas, cysticerci and other intracranial tumours or expanding lesions are calcified with varying degrees of frequency and intensity. Tumours of the brain are more prone to undergo calcification than are tumours in other parts of the body. Why this should be is not entirely clear, but undoubtedly the slow growth of the lesions contributes to it. Another factor is the prevalence of degeneration of small blood vessels of the tumour and its surroundings, with deposition of calcium in or about the walls of the vessels. Some tumours may even contain bone (Fig. 1).

(d) **Vascular Changes**—When tumours are attached to the dura, the neighbouring meningeal vessels are usually enlarged. Increased vascularity of this type can be inferred from the abnormal appearance of the meningeal grooves in radiograms. It is in meningiomas (dural endotheliomas) that changes of this sort are particularly apt to occur and they constitute an important indication of the nature and situation of tumours that may give few obvious clinical localising signs. In these cases the enlargement of the meningeal arteries presumably occurs in response to the needs of the tumour, just as with tumours in other parts of the body.

The meningeal arteries supply the skull bones as well as the dura, and when one of them is enlarged there is an increase of blood supply to the adjacent bone, as observations at operations show. This also can often be shown by X rays.

(e) **Local Effects of Intracranial Tumours upon the Skull**—Intracranial tumours on the surface of the brain usually produce changes in the overlying bone. These changes are the same as those produced in other bones by adjacent tumours, but they are of much greater importance in radiological diagnosis, since they provide what may often be the only clue to accurate localisation of the tumour. In this connection it is necessary to consider at some length certain fundamental facts of the pathology of bone.

In the normal skeleton new bone is constantly being formed (*deposition*) and old bone is constantly being removed (*resorption*). In this way the skeleton changes its shape and size according to the needs of growth. In the skull, which is partly formed in membrane and partly in cartilage, these changes are constantly taking place, with the normal growth of the brain, resorption predominates in the inner table and deposition in the outer table. When the skull is affected by a pathological condition the balance between these two processes may be disturbed, increased deposition and diminished resorption result in thickening (*hyperostosis*) or increased density (*osteosclerosis*) of the

bone, increased resorption and diminished deposition result in thinning or diminished density of the bone (*osteoporosis*). The term 'expansion of bone' is frequently used. Such expansion is not due to stretching of bone but is the result of resorption of bone on the surface exposed to the pressure, and simultaneous deposition of bone on the other.

All radiological changes in the skull itself as distinct from changes in its blood vessels are to be explained by these processes. The work of the morbid anatomists especially *Pommer*, *Schmorl* and *Turnbull* has clearly shown that there is no absorption or disposition of calcium independent of the removal and formation of bone. Thus prolonged pressure of a tumour against the inner table of the skull may produce diminished density of the bone as seen in radiograms and thinning or even perforation. This is not due to the abstraction of calcium from the bone but to resorption by macrophages (*osteoclasts*) of bone with its calcium. It is reasonably certain that *hyperostosis* and *osteosclerosis* on the one hand and *osteoporosis* on the other are produced solely by the activity respectively of *osteoblasts* and *osteoclasts*.

It might be thought that a tumour invading the skull would invariably excite *osteoclasts* to destroy it but that is not so. In some cases the tumour laden bone shows a scanty bone content and diminished density in radiograms but in others the amount of bone is increased and the density of the part as

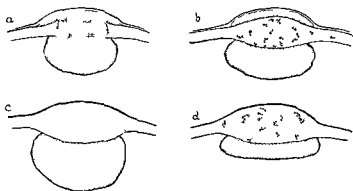


FIG. 26.—Varieties of reaction of the skull to invasive tumours

seen radiologically is excessive. The rapidity with which the invasion occurs is not a sure guide though it is often true that the more rapid the invasion the greater the *osteoporosis*. As a general rule bone around a tumour behaves like any other connective tissue under similar conditions: the tumour which in soft tissues calls forth great deposition of fibrous tissue is in bone *osteosclerotic* while the tumour which in soft tissues excites little or no chronic inflammatory reaction is in bone *osteoporotic*.

THE REACTION OF THE SKULL TO INVASIVE TUMOURS is shown diagrammatically in Fig. 26. It will be seen that (a) the bone may be almost

completely resorbed; (b) it may be widened, but at the same time show osteoporosis; (c) it may be widened, but increased in density owing to great deposition of new bone (osteosclerosis); and (d) it may show in places excessive resorption and in others excessive deposition. None of these changes is specific to any particular type of tumour, a fact to which *Taylor* has drawn attention. The slow-growing meningioma, with relatively feeble power of invasion,<sup>1</sup> will in one case produce an osteoporotic reaction, and in another an osteosclerotic reaction; the metastatic carcinoma of the skull will likewise produce varying reactions. These facts, which have perhaps not been sufficiently appreciated in the past by radiologists, inevitably limit the precision of radiology in pathological as opposed to regional diagnosis.

THE REACTION OF THE SKULL TO NON-INVASIVE TUMOURS is shown diagrammatically in Fig. 27. It will be seen that: (a) the bone may be thinned, with or without local bulging, or completely resorbed; (b) the bone, though not thinned in its contours, may be much less dense, the trabeculae are scanty

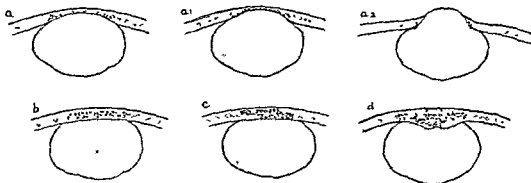


FIG. 27—Varieties of reaction of the skull to non-invasive intracranial tumours

and the medullary spaces are occupied by fibrous tissue; (c) the bone may be for the most part affected as in (b), but there are in some places areas of increased deposition; (d) the bone may project internally, producing an *internal hyperostosis*<sup>2</sup>; although the bone is thickened, yet it is in most places less dense than normal, the bony trabeculae having been extensively resorbed and largely replaced by fibrous tissues, or by an unusually large number of blood-vessels. In such cases, however, there are usually one or more areas of osteosclerosis. Localised or circumscribed hyperostosis is seen particularly in meningiomas, and Fig. 28 shows the actual histological appearance of one example. It

<sup>1</sup> Many meningiomas are encapsulated, and not invasive, but a certain number of them do spread into the medullary spaces of the overlying bone. Such tumours must be regarded in the pathological sense as invasive tumours, even though the majority of them are clinically benign, and, apart from their invasive property, histologically benign.

<sup>2</sup> The term *Hyperostosis*, as applied to the calvarium, indicates a generalised or localised increase in volume of the skull, due usually to invasion of the bone by tumour, but sometimes to inflammation, to traction, or possibly, as in diffuse frontal hyperostosis, to endocrine disturbances. Localised hyperostosis may be external, or internal, or may project from both inner and outer tables of the skull.

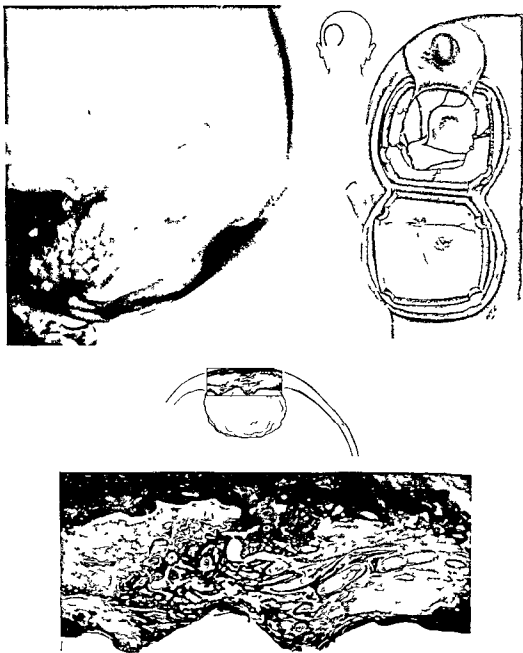


FIG 28 —Left occipital meningioma with internal hyperostosis The microphotograph of the hyperostosis shows fibrosis of the medullary spaces with scanty trabeculae of coarse fibred bone The hyperostosis does not contain tumour tissue Case No 112



may well be that, as a result of formation of collagenous fibrous tissue in their substance, meningiomas at some time in their growth exert traction on the overlying skull, and that this is responsible for the production of hyperostosis.

Without radiograms that show contours, it is difficult at times to determine whether an area of altered density in the skull is associated with thickening or thinning of the bone, or alteration of its contours. For this reason all such areas should be examined by tangential projections.

It will be seen that by means of radiograms we can in certain cases tell the situation and even the nature of the lesion. In other cases, it is possible to say that there is rise of intracranial pressure, due to tumour or other expanding lesion, but there is no evidence as to the situation and nature of the lesion. In some cases the absence of any radiological signs may constitute information of value in diagnosis.

## CHAPTER II

### THE RADIOLOGICAL FEATURES OF THE CRANIUM, NORMAL AND PATHOLOGICAL

THIS CHAPTER deals with systematic radiological examination of the cranium, and draws attention briefly to the normal and abnormal appearances that are commonly found. A systematic description of the abnormalities is given in a later chapter. In this chapter they are considered from the point of view of differential radiological diagnosis. In examining radiograms of the skull it is useful to have a system. The one described here is based on examination in sequence of the following points:

- (1) The size, shape, and contours of the skull
- (2) The sutures
- (3) The cranial vault, its thickness and texture, the presence of local areas of increased or diminished density, thickening or thinning, the presence and extent of convolutional markings
- (4) The vascular channels (a) meningeal channels, (b) diploic channels, (c) sinus markings
- (5) The sella turcica
- (6) The base of the skull, especially the lesser wing of the sphenoid and the petrous bone. In certain cases it may be necessary to study the optic foramina, internal auditory meatuses, the foramen magnum, and other basal foramina
- (7) The presence and position of calcification, normal or abnormal
- (8) The presence of air within the cranium (aerocoele)
- (9) In addition a brief examination of the accessory sinuses is made for sometimes abnormality in one or other of these will lead the observer to the correct solution of problems of intracranial diagnosis

#### THE SIZE AND SHAPE OF THE SKULL

The vault of the skull is comparatively large at birth and grows rapidly, so that it has almost reached full size by the time the child is 6 or 7. The rest of the skull grows more slowly, and continues to increase in size until the age of 20. Great variations occur in the size and shape of the normal skull, for details of which the reader is referred to anatomical treatises and to the monograph of *Schuller*. Deviations from the normal size and shape may or may not be associated with underlying disease and diagnosis of intracranial abnormality should rest on clinical examination rather than on radiological appearances.

Of abnormalities in the size of the skull as a whole, enlargement is the more common. When the bone is not thickened the cause is almost without exception hydrocephalus, when the bone is thickened the cause of the enlargement must be sought among such disorders of bone growth as osteitis deformans, osteitis fibrosa, familial osteosclerosis, and acromegaly. Smallness of the head may be associated with open or closed sutures. With open sutures it is usually due to failure of development of the brain (microcephalus). With closed sutures it is due to craniostenosis.

Alterations in shape of the base of the skull are found in some cases of hydrocephalus, the atlas and axis pressing into the posterior fossa. This condition is known as *basilar invagination*. Localised alterations in the shape of the vault are considered in the following pages.

### THE SUTURES

The exact dates of closing of sutures of the vault are rarely of importance in radiological diagnosis because they are subject to such great variations. Most of the sutures of the base are joined at birth,<sup>1</sup> while those of the vault do not close fully until after the age of 35, or even considerably later. Small supernumerary bones, called Wormian bones, are sometimes seen in the vault, especially in the region of the lambdoid suture. They arise from separate areas of ossification and are of no definite significance in diagnosis, though they indicate some abnormality of ossification.

Separation of all the sutures of the vault occurs in hydrocephalus from any cause, up to the age of 15 (Fig. 23). Later in life local separation of one suture may indicate a subjacent tumour of slow growth. Premature closure of the sutures is found in craniostenosis, delayed closure occurs in rickets and other deficiency diseases, renal infantilism, cleido cranio dysostosis, and fragilis osium.

### THE CRANIAL VAULT

#### A. Normal

The bones of the skull arise in two different ways—those of the vault from membrane bone, and those of the base and face from cartilage. Up to the age of 6–7 the bones of the vault appear as one layer in a radiogram, but after that differentiation begins and three layers are to be seen, the inner and outer tables of compact bone, and the diploë of cancellous tissue lying between. In old age these layers tend to disappear once more, so that the vault again becomes uniform in density, at the same time the whole skull appears more translucent, except in the frontal region, where it becomes thickened, presumably as a sequel to atrophy of the frontal lobes of the brain.

Skulls vary greatly in their thickness. From cranial operations it is found that thin skulls may be very hard and contain little diploë tissue, while thick

<sup>1</sup>The basi occiput and basi phenoid do not unite until puberty or later.

skulls are sometimes soft. The significance of these differences is not known. Some parts of the cranial vault are much thinner than others especially the parts covered by muscle. The thinnest of them all is the squamous temporal bone<sup>1</sup> which in consequence gives an area of diminished density in lateral radiograms. A second area of diminished density is seen in the occipital region in true lateral radiograms and sometimes a third at the frontal pole. These areas tend to disappear when the head is rotated slightly. They arise probably because the amount of brain tissue is small in relation to the capacity of that part of the skull. They are not due to any local thinning of the bone.

Small localised areas of thinning of the skull appearing in the dried skull as indentations of the inner table on each side of the superior longitudinal sinus are often seen in radiograms as circumscribed areas of diminished density. They are produced by localised dilatation of the cerebral veins entering the superior longitudinal sinus (sinusoidal veins) or by Pacchionian granulations and they do not give rise to symptoms. The venous depression has a sloping edge while the depression caused by Pacchionian granulations is sharply punched out. Similar localised erosions of the inner table of the frontal bone at some distance from the middle line are frequently seen in postero-anterior radiograms (Fig. 3). They are due to arachnoid granulations which are identical in structure with the parasagittal Pacchionian granulations and they do not indicate a pathological lesion though it is to be noted that they are apt to be excessive in chronic non-suppurative leptomeningitis.

The areas of diminished density so far described can be distinguished as a rule from areas of diminished density due to meningioma or other lesions by the following points: (1) the absence of any bony projection from either surface of the skull; (2) the absence of abnormal vascular markings in the neighbourhood; and (3) though this is not invariable their symmetrical distribution. The radiological evidence however is not always decisive and when clinical evidence indicates unequivocal disease of the subjacent brain the temptation to make a diagnosis of tumour is strong. In all cases of doubt air injection should be employed.

Symmetrically placed circular translucent areas are sometimes found in the parietal bones as a congenital defect.

The porosity of the cranial vault varies greatly. Sometimes particularly in young adults the vault appears to be honeycombed by large and small spaces which correspond to the medullary spaces of the diploe. This appearance is usually of no pathological significance though occasionally it may be the first sign of generalised osteoporosis.

**Convolutional Markings**—In children the inner table of the skull lying

<sup>1</sup> The tabular part of the occipital bone below the superior curved line is also very thin except in the middle line.

The illustrations are indebted to Miss Audrey J. Arnott or Fig. 96 and of a few sketches in Part I.



FIG 23a—Thinning of parietal bone due to a large underlying astrocytoma in a female aged 24 years. Note the changes in the sella turcica

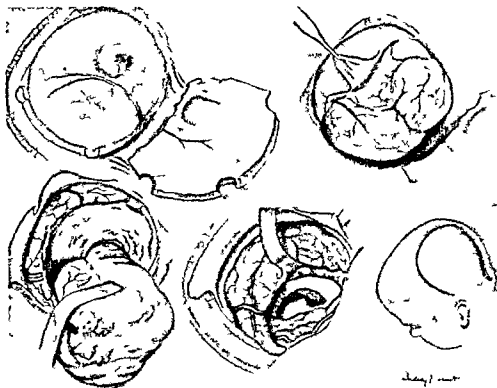


FIG 23b—The same case. At operation the bone was extremely thin, one area posteriorly was no thicker than paper and was bulging. Around this the bone was opaque and white corresponding to the area of diminished density in the radiogram. In this white area the meningeal channel was quite deep, but did not show in the radiogram. Case No. 2874

above the tentorium cerebelli normally presents shallow depressions corresponding in size and direction with the convolutions of the cerebral hemispheres. Between these depressions are ridges of thicker bone which correspond to the cerebral sulci. These convolutional markings are readily seen in the vault in radiograms. Convolutional markings in adults are usually a sign of raised intracranial pressure but up to the age of 15 they are normally present in parts of the vault. In children therefore their presence should not be regarded as evidence of a pathological process unless they are extensive when there is often also separation of the sutures. In lateral radiograms lines or areas of thick bone between the convolutional depressions of the squamous temporal bone may cross the shadow of the sella turcica and have sometimes been mistaken for calcification of a suprapituitary tumour. An observer with good stereoscopic vision will usually be able at once to localise such areas

of increased density to the lateral wall of the skull but if this is difficult the question can be settled by parallax.

## B Pathological

### Diminished Density of the Skull

—Areas of diminished density of the vault of the skull which are abnormal in situation and extent are due to thinning from pressure atrophy or to replacement of bone by soft tissue. The generalised thinning which gives rise to the appearance of increased convolutional markings has already been mentioned (Fig 24). It indicates a rise of intracranial pressure and gives no clue to the situation of the lesion.

Localised thinning is sometimes found over the summit of slow growing superficial tumours and may be accompanied by slight projection of the thin part of the vault (Fig 25). In radiograms it is seldom possible to tell whether

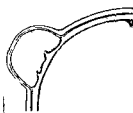


FIG 30 Localised erosion of the skull due to subdural dermoid tumour in a man aged 24. Diagram of the same tumour from information obtained at operation Case No 3148.

the bone has been completely destroyed or whether it persists as a thin shell. The latter is the more usual finding at operation. Of the tumours that produce thinning of the vault slow growing gliomas, especially astrocytoma, are the

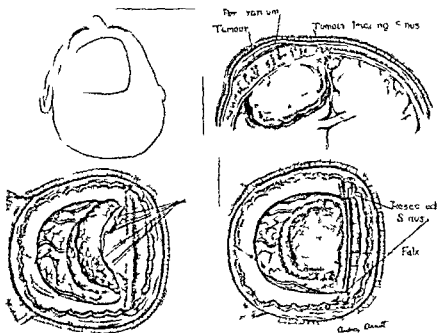


Fig. 31 — Parasagittal meningeoma with hyperostosis. The sketches of operation show that the intracranial part of the meningeoma is larger than the hyperostosis and that it has invaded the sigmoid sinus. Man aged 60. Case No. 779.

common (Fig. 29). Cholesteatoma of the vault, hydatid cyst, and circumscribed superficial tumours of slow growth may also produce the effect. It is rare in meningeoma. Localised thinning of the skull is also caused by pressure from without by benign tumours of the scalp and cranium such as dermoid and epidermoid tumours (Fig. 30). When bone is replaced by soft tissue either neoplastic or inflammatory, an

appearance of diminished density results. In these circumstances the bone is usually thickened. Benign tumours limited to the skull may also produce such areas of diminished density e.g. hæmangioblastoma of the skull (Fig 163). The tumours which by invasion of the skull, produce areas of diminished density include *meningioma*, metastatic tumours, *myelomatosis*, and *carcinoma* or *sarcoma* invading the skull from without (e.g. rodent ulcer). Fig 37 shows a *meningioma* with bony projection which in the radiograms appeared as an area of diminished density. The tumour excited an osteoporotic reaction; it has already been remarked that *meningiomas* more commonly produce an osteosclerotic reaction. Reduction of the density of



FIG 37—Left frontal *meningioma* with hyperostosis in a woman age 152. The swelling had been present for four years growing slowly. There was a scar of old injury in the scalp over the lump. Case No. 2691.



the bone over a *meningioma* is not always due to invasion of the bone by the tumour. Fig 28 shows an area of diminished density over a small occipital *meningioma* which was entirely due to fibrosis of the marrow. The tumour had not invaded the bone at all.

Bone may be replaced by granulation tissue in pyogenic osteomyelitis or by granulomatous tissue in syphilitic or tuberculous caries. Diminished density of the skull is a conspicuous feature of xanthomatosis of the skull.

**Increased Density of the Skull**—Increased density is usually associated with thickening of the skull. With pathological thickening the radiologic distinction between the diploe and the inner and outer tables is usually lost. Generalised increase of density is found in Paget's osteitis deformans in mar-



very slow growth, and at times cannot be distinguished either clinically or radiologically from meningioma (Fig 165)

(3) **Osteitis Fibrosa**—This is the most common pathological lesion that produces the condition known as leontiasis ossea. Osteitis fibrosa affects the

base more often than the vault, but occasionally will produce a bony swelling in the temple or forehead, usually as an extension of a basal lesion. It is frequently associated with pathological bony changes elsewhere especially in the facial bones.

(4) **Osteitis Deformans**—When it affects the skull osteitis deformans is usually diffuse but occasionally it forms a localised swelling. Fig 34 shows such a case.

(5) **Osteoma of the Skull**—This can usually be recognised radiologically by its extreme density and sharp outline in tangential views. Multiple osteomas occur at times as in hemi-craniosis. The bony swelling in this type of tumour is usually small. The radiological appearances which are often distinctive are described in Chapter IV.

(6) **Cavernous Hæmangioma of the Skull** (Fig 163) See Chapter IV.

(7) **Sarcoma of the Skull**—The distinction of this lesion from other bony lumps rests in the first instance upon its more rapid growth and secondly upon its poor density to X rays. Indeed even a very large lump may not contain enough bone to show in a radiogram of standard penetration (Figs 3 and 36).



FIG. 34—Osteitis deformans confined to one side of the cranial vault (confirmed histologically). Man aged 44. Bony prominence of right side of head of seventeen years' duration. Also prominence of left superior maxilla for a similar period. Case No. 2611.

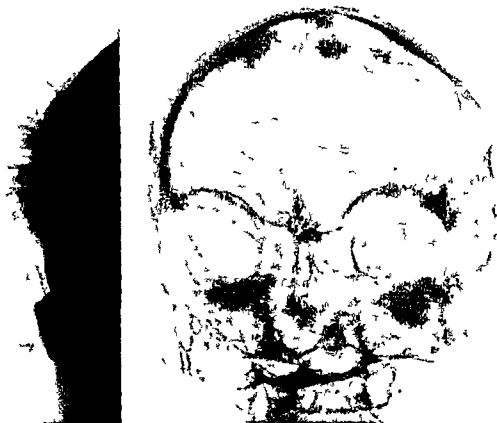


FIG. 35—Sarcoma of right temporal bone. In the normal exposure only slight thickening of the bone is seen. The deposition of bone in the tumour in the characteristic form of radiating spicules is only seen when very soft radiation is employed. Case No. L.H. 22058/1935.

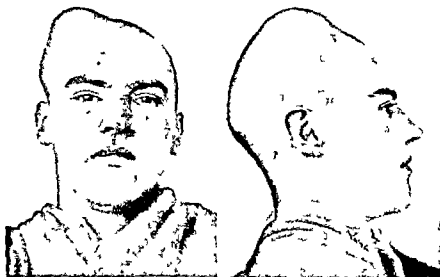


FIG. 36—Another case of sarcoma of skull. In spite of the large size of the lump the radiogram showed practically no calcification. Case No. 1625.

## B The Bone is Thinned

(1) **Intracranial Tumour**—Bony lumps due to intracranial tumour covered by thinned bone are usually inconspicuous and are often not found until X ray examination has been done or the head shaved. They are most commonly associated with astrocytoma, the most slowly growing of the gliomas (Fig 2a) and with cholesteatomas and are met most often in children or young adults. We have found such bony projections in the temporal parietal

occipital and suboccipital regions. The bone is so thin that it may not show unless under exposed tangential projections are taken.

Meningioma on rare occasions may produce thinning and expansion of a small portion of the overlying skull.

(2) **Hodgkin's Disease** Lympho sarcoma Lymphatic Leukæmia Xanthomatosis—In these conditions the skull is thinned to greater or less degree but is not expanded. The swelling is due to the presence of new tissue between pericranium and bone.

In xanthomatosis



FIG. 3. Thinning and projection of right squamous temporal bone present since birth in a man aged 20 who complained of headache of five weeks duration. At operation an old and recent subdural hæmatoma was found on the right side associated with an arterial abnormality of the underlying cortex. Case No. 459.

In xanthomatosis the swelling is most common in the early stage of the disease. After X ray treatment the swelling is usually replaced by a depression.

(3) **Subdural Hæmatoma**—When it occurs in infancy and is left untreated subdural hæmatoma may produce local thinning and bulging of the skull (Fig 3b).

## VASCULAR CHANNELS

The brain is supplied by the internal carotid and vertebral arteries, the meninges by the middle meningeal branch of the internal maxillary artery.

and by twigs from the other arteries which go to the cranium. The venous return is more complicated. Cerebral veins, taking a course that is quite independent of the cerebral arteries, open into the great venous sinuses. The meningeal veins usually accompany the meningeal arteries and are quite small, they connect also at the middle line with the sagittal sinus by means of *lacunæ laterales*, wide channels in the dura which lie on either side of the sagittal sinus and into which Pacchionian granulations project. There are free communications between many of the cerebral veins and the meningeal veins through the *lacunæ laterales*.

There is a third system of veins, the diploic veins which run in the cancellous tissue of the skull. They are the nutrient veins of the skull, and they become engorged when intracranial pressure is raised, since they communicate freely with the meningeal veins. They are drained by numerous small emissary veins.

Most of the blood leaves the cranium by means of the internal jugular veins, but there are also numerous other emissary veins passing through the named foramina of the base of the skull to the pterygoid plexus, and others passing from the great venous sinuses of the vault out into the scalp. When anything interferes with the return of blood along the internal jugular veins to the heart, as in a fit of coughing, blood escapes from the cranial cavity by way of the emissary veins, and this accounts for the great venous engorgement of the scalp that accompanies prolonged coughing. When intracranial pressure is raised, intracranial venous pressure is also raised, in these circumstances there is great engorgement of the emissary veins and of the diploic veins, as is commonly observed during intracranial operations for tumours and other expanding lesions which raise intracranial pressure. It is to be expected, therefore, that emissary veins and diploic channels will become enlarged as a result of sustained rise of intracranial pressure, and this does occur.

The increase in size of the meningeal arteries that accompanies the growth of a meningeal tumour has already been commented upon in the earlier chapter.

### Normal Radiographic Appearances

For a detailed account of the normal radiographic appearances of the cranial blood vessels the reader is referred to the monograph of *Landblom*.

**The Cerebral Arteries**—The carotid canal, through which the internal carotid artery enters the skull, can often be seen in the submento vertical projection (Fig. 14). The foramen in the transverse process of the axis through which the vertebral artery passes on its way to the cranium, can usually be seen in the ordinary lateral projection of the skull. None of the smaller cerebral arteries when normal produces any radiographic sign.

**The Cerebral Veins**—The cerebral veins mark the skull only in places where they are adherent to or embedded in the dura. This occurs to a

variable degree alongside the sagittal sinus especially near the coronal plane. The veins at this level are often dilated (sinusoid veins) and the radiographic appearance is not so much that of a linear groove as of an irregularly oval but sharply defined area of diminished density on one or both sides of the sagittal suture which corresponds to a depression of the inner table of the skull. It can usually be distinguished from depression due to an underlying tumour by the absence of any projection of the outer table of the skull and by the absence of radiographic signs of abnormal vascularity in the surrounding skull.

**The Dural Sinuses**—The sagittal transverse and sigmoid sinuses often mark the skull (Fig 12) especially in children. There is no constancy about this and the size of the groove is no evidence of the size of the sinus. One transverse sinus may mark the skull while the other does not.

**The Middle Meningeal Vessels**—The middle meningeal artery enters the skull at the foramen spinosum. This foramen can be seen in the submento-vertical projection (Fig 14) immediately posterior and lateral to the foramen ovale; rarely it is absent. From the foramen spinosum the artery passes in a groove across the middle fossa for a short distance and then divides into anterior and posterior branches. The anterior branch usually the larger ascends to the pterion in which region it lies in a deep groove or even buried in the bone (Fig 7). It passes upwards towards the vertex in a groove in the inner table usually a short distance behind the coronal suture. The Rolandic fissure lies approximately 1.2 cm behind this groove which thus provides a useful rough topographical marking in lateral radiograms. The posterior branch lies in a groove passing obliquely backwards over the squamous bone and the posterior part of the parietal bone. Both arteries branch dichotomously. The grooves in which they lie become progressively smaller as the arteries ascend towards the middle line. In the normal skull the grooves of the smaller branches cannot be demonstrated radiologically. The grooves increase in depth up to middle age; they are not usually visible before the age of 2. In the proximal part of their course they are often tortuous. Sometimes the edges of a groove present thin lines of increased density. The arteries of the two sides are usually symmetrical in distribution.

The meningeal veins that accompany the artery are usually small throughout the greater part of their course. Occasionally however a vein accompanying the anterior branch of the middle meningeal artery may be very large. It lies in a deep groove which stands out prominently in lateral radiograms. This groove passes straight from the lesser wing of the sphenoid to the sagittal suture; it is of fairly uniform width and does not branch like the grooves of the meningeal arteries. It may be present on one or both sides and is of no pathological significance. The large vein it contains may be looked upon as an additional dural sinus (*spheno-parietal sinus* or *bregmatic vein*).

**Diploic Channels**—It is not common to see diploic channels in the radio-

grams of subjects below the age of 10. As age advances the diploic veins enlarge though in some people they do not appear at all. They are easily seen in radiograms. Their position in the vault and their size vary greatly but three main sets of veins have been described (*Breschet, Jefferson, and Stewart*)—frontal, parietal and occipital—of which the parietal are those most commonly found (Fig 38). In radiograms diploic veins are seen as wide channels of irregular course and width. In contradistinction to the meningeal channels with their upward and backward direction their direc-



FIG 38—Diploic channels

tionous branching and fairly constant position diploic veins run in any direction and branch and anastomose with one another in star shaped or spiderly fashion. The outline is not sharp and has on it small dilatations which give it a beaded appearance. Diploic channels communicate freely with one another even across suture lines.

Meningeal markings and more rarely diploic markings are sometimes mistaken for fissured fractures. A fracture gives a blacker and more sharply defined shadow since it involves the whole thickness of the skull wall. Fissured fractures do not often branch or cause the appearance of an island of normal bone like that produced by vascular markings unless there is simultaneous depression of the bone. Meningeal markings are symmetrical as can

be shown from lateral stereoscopic projections taken both from the right and from the left

### Pathological Radiographic Appearances

In vascular intracranial tumours the vessels of the overlying scalp also are often enlarged even though the tumour does not invade the bone. Enlarged pericranial vessels often produce grooves on the outer table of the skull but these are rarely large enough to be demonstrable by radiography



FIG. 39—Huge diploic channels in the left frontal region of a man aged 60 with signs of a left frontal expanding lesion (not verified) and a cranial bruit. Presumptive diagnosis: intracranial arteriovenous aneurysm. The arrows point to a ring in the flattened left anterior horn. Case No. 9804.

When intracranial pressure has been raised for any considerable length of time as with tumours of slow growth there is usually enlargement of the emissary veins. This is most easily demonstrated by means of antero-posterior projections in the region of the occipital protuberance where emissary veins from the torcular Herophili perforate the skull. The dilatation of these emissary channels is of no localising significance and indicates only that there is rise of intracranial pressure.

No clear indications for the diagnosis of intracranial lesions can be obtained from radiographic examination of the markings of the sinuses and diploic

veins. It has been claimed by *Schuller*, and by *Elsberg* and *Schwartz*, that the diploic channels are enlarged in the neighbourhood of meningiomas on the convexity of the brain. It is certainly true that exceptionally large diploic channels may occasionally be seen where there is an underlying meningioma, and it is quite likely that their unusual dimensions may be the direct result of raised venous pressure in the region of the tumour, especially if this begins in early life, but it is a matter of common experience that very large diploic channels may exist where there is no underlying tumour. The only safe rule is to place no diagnostic significance on the appearance of diploic



FIG 40.—Right frontal meningioma in woman of 39. Dilated meningeal channels converging on internal hyperostosis, in which are numerous vascular perforations. Changes due to the rise of intracranial pressure are seen in the sella turcica. Case No. 343.

channels, save in the most exceptional appearances (Fig 39), or where successive radiological observations of a case show progressive dilatation.

The markings of the meningeal vessels, unlike those of the diploic channels, may be of signal importance as an indication of the presence of tumours. Dilatation of the foramen spinosum, or increased breadth, depth, and extent of meningeal markings on one side of the skull indicate almost invariably that there is a vascular tumour nearby, usually a meningioma, but sometimes a glioma attached to the dura, or an arterio-venous aneurysm of the cerebral vessels. The relative size of the meningeal channels on the two sides should not be compared in the same pair of lateral stereoscopic films, but rather in lateral projections of both sides of the skull. As the tumour grows, not only do the meningeal channels enlarge but they also become tortuous. At the site of the tumour they give off very numerous fine branches which



can be seen in radiograms as lines, or, when the vessels perforate the bone, as punctate areas of diminished density (Fig 40) There are usually other changes also in the neighbouring bone, such as erosion, or hyperostosis

The dilatation and tortuosity of meningeal channels afford a particularly valuable radiographic sign of the presence, situation, and nature of an intra cranial tumour for these changes occur in just those cases where the clinical signs may present a most difficult problem of diagnosis, namely, in slowly growing meningiomas Such evidence in the radiograms serves also to warn the surgeon that he must be prepared at operation for a lesion of extreme vascularity

### THE SELLA TURCICA

By far the most important part of the base of the skull in radiology is the sella turcica It projects upwards in a way that renders it easily visible in radiograms Many intracranial tumours are situated within or near it, and deform it in notable fashion, and tumours at a distance also readily produce changes in it



FIG 41—Photograph of the middle fossa taken from above the skull to show the relative positions of the anterior and posterior clinoid processes

From the radiological aspect certain anatomical features of the sella are important Firstly, the fossa has comparatively little resistance at the sides, for bone is found only in the anterior and posterior faces and in the floor, so that the pituitary body can expand laterally to some extent before enough pressure is exerted to influence its bony outline Secondly, the anterior clinoid processes are situated much farther from the middle line than

the posterior (Figs 11 and 41), and do not form part of the roof of the sella at all, they are in fact almost out of reach of any but asymmetrically placed or very large intrasellar tumours and this is why erosion of the anterior clinoid processes is relatively uncommon The sphenoidal air spaces lie under the floor of the sella turcica, and this region is consequently weaker in structure than the rest of the bony wall Whether the floor is depressed as a whole or in part when the fossa becomes enlarged depends on how far back the air spaces extend

#### Normal

The size and shape of the sella are seen best in lateral projection It is important that this should be a true lateral view, with rotation of the head about the vertical plane a false impression may be obtained about the dorsum and the size of the sella, with rotation about the antero posterior plane the floor of the sella may be obscured by the shadow of one middle fossa, or of one temporo mandibular joint The anterior clinoid processes (Fig 11) are

also well seen in the antero-posterior view with 25 degrees tilt; the dorsum sellæ and the posterior clinoid processes can be seen either in this view or in the picture taken with 35 degrees tilt (Fig. 12).

The normal sella turcica has many variations in contour, but most of these fall into three groups—circular, oval, and flat (Fig. 42). The average antero-posterior measurement of the sella at its upper margin is a fraction over 1 cm. (variations 0.7–1.2 cm.), and its average depth is 0.8 cm. (variations 0.5–1.1 cm.). The variations are wide, and it is often very difficult, if not impossible, to say from measurements alone that the sella is enlarged.

The shape of the clinoid processes and the height and thickness of the dorsum sellæ also vary greatly. Sometimes the sphenoidal sinus extends into the dorsum sellæ. The anterior clinoid process on each side is united to the posterior clinoid process by ligaments, and in 5–10 per cent. of normal people of all ages these ligaments are partly or wholly ossified on one or both sides, giving an appearance in a radiogram that the sella is bridged above by bone (Fig. 43). But the



FIG. 42.—Variations in the shape of the normal sella turcica.

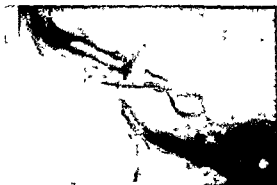


FIG. 43.—"Bridged" sella turcica. Woman, aged 32. Case No. 1295.



FIG. 44.—Ossified petro-clinoid ligament. Man, aged 30. Case No. 1313.

apparent bony bridge is only in the interclinoid ligaments, and not in the diaphragma sellæ.

Ossified ligaments are rarely seen running between the anterior and middle, or middle and posterior clinoid processes. The posterior clinoid process is connected to the apex of the petrous bone by a ligamentous fold of dura, known as the petro-clinoid ligament, which is occasionally ossified (Fig. 44). A fold of dura that runs from the posterior limit of the middle fossa to the anterior clinoid process is also occasionally ossified.

### Pathological

Smallness of the sella appears to be but a variation of the normal, apart from rare cases where there is pathological thickening of its bony walls. In spite of the claim of some clinical endocrinologists to the contrary, there is as yet no definite evidence that smallness of the sella indicates under development of the pituitary body.

Enlargement of the sella is produced by intrasellar tumours and aneurysms or by tumours and other expanding intracranial lesions at a distance. No



FIG 45—Enlargement of the sella turcica in a case of chromophobe pituitary adenoma. Woman, aged 42. The dorsum sellæ is eroded and the posterior clinoid processes appear to be isolated. Case No. 949.

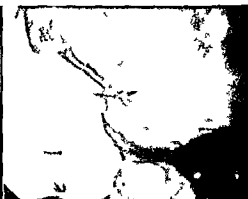


FIG 46—Case of large chromophobe adenoma with right temporal intracranial extension. Woman, aged 48. The sella turcica has been grossly dilated and eroded and only the anterior clinoid processes can be clearly seen of these the right is eroded and displaced upwards. Case No. 2176.

only is the sella enlarged, but its shape is altered, and there is usually obvious destruction also of some part of its bony wall.

Intrasellar tumours include pituitary adenomas, epidermoid tumours of the hypophyseal stalk (cranio-pharyngeal tumours), gliomas of the optic chiasm, and cholesteatomas. As the tumour increases in size the floor of the sella becomes thin and depressed, even below the level of the middle fossa, whereas normally it lies about 1.5 cm. above the middle fossa. The dorsum sellæ is displaced backwards, sometimes the posterior clinoid processes are also displaced backwards, but at other times the interclinoid ligaments are evidently strong enough to prevent this occurring, and the sella then has a ballooned appearance. The destruction may be complete, or almost complete in the middle of the dorsum, so that the posterior clinoid processes appear isolated (Fig 45). There is at times prolongation of the fossa beneath the anterior clinoid processes.<sup>1</sup> As enlargement proceeds destruction also occurs

<sup>1</sup> In children, forward prolongation of the pituitary fossa is often normal.

and eventually both floor and dorsum sellæ may be reduced to a mere shell which is no longer visible radiographically. In parts of the floor destruction may be complete and the tumour then extends into the sphenoidal sinus

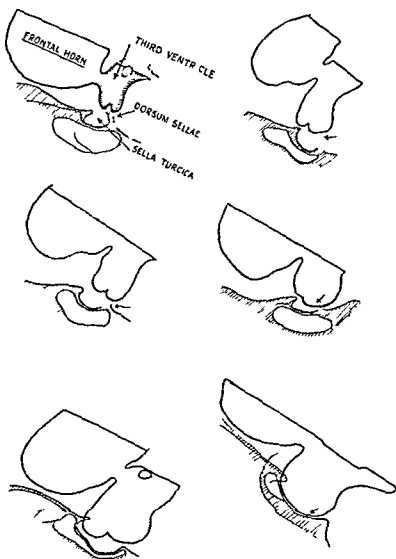
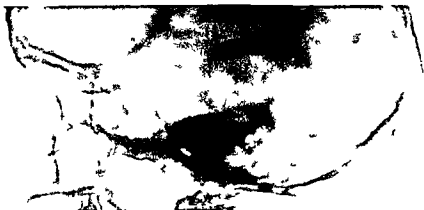


FIG. 47—Stages in the expansion and destruction of the sella turcica by a dilated third ventricle (after Tanning)

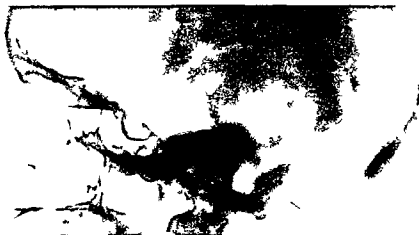
The anterior clinoid processes which are strong and lie outside the fossa, are the last parts to be destroyed, but even they may be splayed out and displaced upwards by the advancing tumour (Fig. 46)

The mechanism by which intracranial tumours at a distance produce enlargement of the sella turcica is readily understood when we recall that the

anterior part of the third ventricle lies immediately above the diaphragma sellæ (Fig 47). Like the lateral ventricles the third ventricle becomes greatly dilated when tumours or other lesions of the posterior fossa or midbrain obstruct the outflow of cerebrospinal fluid. The anterior part of the third



a



b

FIG 48 (a) Enlargement and thinning of the sella turcica with disappearance of the dorsum in a case of cerebellar astrocytoma. Girl aged 17.

(b) Same case five years after removal of tumour showing reappearance of sella. Case No 1.

ventricle then presses down upon the diaphragma sellæ and if that structure is weak or as it often is deficient at its centre considerable pressure can be exerted on the walls of the sella causing them to expand. With long continued pressure the posterior clinoid processes and dorsum sellæ tend to be destroyed from above downwards.

Enlargement of the sella turcica from dilatation of the third ventricle may simulate in radiograms the enlargement caused by pituitary tumour, and moreover may be associated with the appearance of symptoms like adiposity and amenorrhœa which are due to secondary disturbance in the pituitary or suprapituitary regions. Diagnosis between the two types of sellar enlargement may thus be a matter of great difficulty. Fig 48 shows the sella turcica of a girl of 17 who was found at operation to have a cystic and solid astrocytoma of the cerebellar vermis. Before operation this patient had become almost blind from optic atrophy and had had amenorrhœa, and the first radiologist who examined her hazarded a diagnosis of pituitary tumour from the appearance of the sella turcica. Further clinical investigation of this case indicated the correct diagnosis but in some cases it may be extremely difficult to be certain of the primary cause of a sellar enlargement.

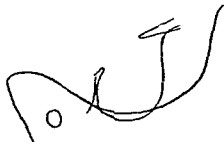


FIG 49—Deepening of the sella turcica due to tumour at a distance (cerebellar vermis astrocytoma). Man, aged 20. Case No 13.

Enlargement of the sella turcica from tumour at a distance is not always

due to dilatation and downward pressure of the third ventricle but may be produced by tumours of the cerebral hemisphere which do not cause dilatation of the third ventricle. This enlargement is in our experience usually only slight. Tumours of the cerebral hemisphere seem to have greater power of destroying the dorsum sellæ than of enlarging the sella. The destruction usually appears as a thinning of the whole dorsum.

Do the radiographic appearances help us to distinguish between the sellar enlargements of pituitary



FIG 50—Asymmetrical enlargement of the sella turcica in a case of chromophobe pituitary adenoma. Man, aged 60. The right side of floor of sella is more expanded and is seen as a thin linear shadow lying below the floor of the left side. This is quite obvious when viewed stereoscopically. Case No 134.

tumours and those of raised intracranial pressure? As a rule the more experienced the radiologist the more cautious he is in interpretation. Very great enlargement of the sella where the antero-posterior diameter and depth are

greater than 2.5 cm. is almost invariably due to pituitary tumours. In sellar enlargement due to generalised rise of intracranial pressure there is not as a rule much increase in the depth of the sella though there is often considerable increase of the antero-posterior diameter but there are exceptions to this rule

(Fig. 49). Irregular enlargement where one side or one portion of the sella is more enlarged than the rest (Fig. 50) is usually due to intrasellar tumour and is explained by the fact that pituitary adenomas may arise in any part of the anterior lobe and sometimes eccentrically. *Hollowing out of the* under surface of one anterior clinoid process also indicates an intrasellar lesion and so do gross resorption and thinning of the floor of the sella though tumours at a dis-



FIG. 51. Enlarged sella turcica in a case of glioma of the optic chiasm. There is a characteristic forward prolongation almost an excavation. Boy aged 5. Case No. 2438.

tance can also produce this. The presence of calcification within or above the sella also indicates a local lesion.

Radiographic appearances alone will not always establish the nature of a sellar enlargement. In each case therefore the problem should be reviewed from the clinical side with especial care. Clinically the difficulty is not so much to separate intrasellar tumours from tumours at a distance such as cerebellar tumours as to distinguish between suprasellar (third ventricle) tumours and cerebellar tumours. Simple radiological examination of the skull often contributes nothing to the elucidation of this problem but ventriculography as we shall see is decisive.



FIG. 52.—Destruction of dorsum sellae without enlargement in two cases of suprasellar epidermoid tumour.

Localised enlargement of the anterior part of the sella turcica occurs sometimes in glioma of the optic chiasm (Fig. 51) when the tumour extends into the optic nerves (*Martin and Cushing*) and rarely also in anteriorly placed pituitary adenomas or in certain types of epidermoid tumour.

Destruction of the sella turcica may occur with or without enlargement. Destruction of the sella with enlargement has already been mentioned. Destruction without enlargement may be produced by suprasellar tumour (Fig 52), but is more often due to general rise of pressure within the cranial cavity, and is thus no indication of the situation of the lesion. The dorsum sellæ and posterior clinoid processes are more readily affected by pressure than the rest of the sella, owing to their unprotected position. The earliest manifestation is thinning of one or both posterior clinoid processes with or without thinning of the dorsum. In long continued pressure the posterior clinoids and dorsum may become completely destroyed. Fig 53 shows a series of cases of tumours of the cerebral hemisphere in which the dorsum sellæ was thinned or destroyed without general enlargement. These changes



FIG 53 — Alterations in the sella turcica produced by tumours of the cerebral hemisphere. Four cases.

will not be visible in radiograms until several months after intracranial pressure becomes raised and they do not occur in every case of rise of pressure, even when it is of long duration. After the rise of intracranial pressure has been relieved by removal of the tumour at operation the dorsum sometimes reappears taking several months to do so (Fig 48). Suprasellar tumours not infrequently produce destruction of the dorsum without enlargement of the sella (Fig 52).

The anterior clinoid processes are not so readily affected by pressure as the posterior, and there is rarely any great change in them radiologically in cases of general rise of intracranial pressure. Schuller claims that they may be thinner and shorter in such conditions but as they vary greatly in size and shape in normal individuals it is not wise to place pathological significance on such appearances when both anterior clinoid processes look the same. Partial destruction of one anterior clinoid process, on the other hand, is an



important early radiological sign for it indicates an expanding lesion nearby. Thus it may be produced by a pituitary adenoma growing eccentrically. We have seen it as an early sign in two cases of chromophobe adenoma: one a solid tumour that projected upwards between the right optic nerve and internal carotid artery and the other a cystic suprasellar chromophobe adenoma. As *Sosman* has pointed out, destruction of one anterior clinoid process is also seen with an aneurysm of the terminal part of the internal carotid artery or its adjacent primary branches (Fig 54). It may be produced also by gloma of one optic nerve. Destruction of the sella is reported by *Schuller* as a result of osteomyelitis, syphilis and tuberculosis of the sphenoid. The floor of the sella may be destroyed by neoplasms of the sphenoidal sinus and by chordoma.

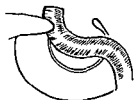


FIG 54—Diagram to show the relationship of the internal carotid artery to the anterior clinoid process.

Thickening of the sella is not common and is usually localised to one anterior clinoid process or to the tuberculum sellae. It signifies as a rule that there is a meningioma nearby arising from the inner part of the lesser wing of the sphenoid or from the posterior part of the olfactory groove. Diffuse thickening of the sella turcica is uncommon. In one case of our series it was associated with a large meningioma of the right middle fossa (Fig 55) and it may also be seen in osteitis fibrosa.

Calcification within or near the sella is of great help in the radiological diagnosis of pathological changes in the sella turcica. The following are the more common causes of such calcification:

(a) Epidermoid tumour of the pituitary or suprapituitary region

(b) Suprasellar meningioma (rarely)

(c) Calcification of the internal carotid artery

(d) Calcification in the wall of an aneurysm of the circle of Willis

(e) Gloma of the third ventricle (rarely)

(f) Pituitary adenoma (very rarely)



FIG 55—Diffuse thickening of the sella turcica and floor of the middle fossa in a case of meningioma of the right middle fossa. The patient, a man aged 21, also had von Recklinghausen's neurofibromatosis. Case No. 564.

Finally, it is important that the radiologist should bear in mind that certain expanding and inflammatory lesions of the pituitary region can give rise to the chiasmal syndrome (optic atrophy and bitemporal hemianopia) without producing any radiological signs of abnormality of the sella turcica. In this group are included certain cases of pituitary adenoma arising above or extending through the diaphragma sellae, suprasellar meningioma, and arachnoiditis of the chiasmal region.

### THE BASE OF THE SKULL

The outline of the anterior, middle, and posterior fossae can be seen in profile in lateral stereoscopic projections (Fig. 7), or in planigrams. The surface and the various foramina of the base of the skull can be studied by fronto-occipital (half axial), submento-vertical (axial), and other special projections (see Chapter I, and Figs. 12 and 13). Pathological changes of radiological significance consist chiefly of thickening or thinning of the bone as seen in profile, and alteration of size of the foramina, and of density of the bone, as seen in the various axial projections. Vascular changes occur, and of these the most important is dilatation of the meningeal vessels, secondary to the growth of a meningioma, with corresponding increase in size in the foramen spinosum as seen in submento-vertical projections. Minor differences in the size of the foramen spinosum on the two sides are within limits of normal, and the foramen may be absent on one side.

In the anterior fossa thickening of the bone between the crista galli and the tuberculum sellae usually denotes the presence of an olfactory groove meningioma, but interpretation should be cautious, for in some normal skulls this part of the base is thicker than the surrounding parts. Thickening of the orbital roof denotes meningioma, secondary carcinoma, or fibro-cystic disease of bone. Thinning of the orbital roof may be due to intracranial tumour, such as cholesteatoma, to orbital tumours, orbital periostitis, or mucocele of the ethmoidal or frontal sinus.

Thickening or thinning of the middle fossa is due to intracranial tumours, or to general bone disease. As a general rule, the tumours that cause thickening of bone are meningiomas or secondary carcinomas; the tumours that cause thinning of the bone are cholesteatomas and other tumours containing structures resembling dermis and epidermis, or temporal gliomas of very slow growth. Inflammatory cysts of the sphenoidal sinus also by their expansion may cause thinning of the middle fossa.

The posterior fossa is very thick in the middle line between the occipital protuberance and the foramen magnum, even up to 2 cm. in width. Between the middle line and the mastoid process it may be very thin, as can be seen in stereoscopic antero-posterior projections. Pathological changes seen in radiograms are few. The presence and significance of foramina of emissary veins below the external occipital protuberance have already been described.

Rarely there is thinning and bulging of one side of the posterior fossa, due underlying tumour of slow growth, but it is important to note that appearance may be produced in lateral projections of some normal skulls slight lateral inclination and rotation of the head. Destruction of part the posterior fossa by intracranial tumour is occasionally seen.

The regions of the base of the skull that are of particular importance radiologically are (1) the lesser wing of the sphenoid (2) the sphenoid fissure and optic foramen, (3) the petrous bone and internal auditory meatus and (4) the mastoid process.

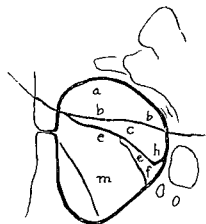


FIG. 56.—Diagram of the normal right orbit and lesser wing of the sphenoid as seen in the postero-anterior projection (a) Roof of orbit (b) Anterior margin of lesser wing of sphenoid (c) Lesser wing of sphenoid (d) Posterior margin of lesser wing of sphenoid (e) Sphenoidal fissure (f) Anterior clinoid process (g) Greater wing of sphenoid

The apex of the triangle is situated laterally, where the lesser wing joins with the temporal portion of the greater wing. The sphenoidal fissure lies below the upper half of the lesser wing and is usually shaped like an inverted comma. Below and external to it can be seen a considerable part of the greater wing of the sphenoid bounded externally by a line of increased density running downwards and medially across the outer part of the orbit. This line is the profile view of the edge of the greater wing.

The lesser wing shows more prominently than the other structures seen through the orbit because of its greater density. Sometimes it is extremely dense and prominent especially in subjects whose cranial vault is very thick. As David has pointed out asymmetry of the lesser wings in thickness and density may be occasionally met with in normal subjects.

### The Lesser Wing of the Sphenoid

**Normal**—The lesser wing of the sphenoid sometimes called the sphenoidal ridge is best studied by postero-anterior projections which as described in Chapter I, the tube tilted 20 degrees towards the vertex from the long axis of the head (Fig. 9). Without the tilt the lesser wing shadow overlaps that of the petrous bone, and one of the most important features of this projection is obscured. The lesser wing of the sphenoid is seen passing transversely across the orbit as a rough triangular area of greater density than the surrounding parts (Fig. 56). Its narrow base lies medially and is limited by the projection of the inner wall of the orbit. The lower border of the triangle corresponds to the posterior edge of the lesser wing, it is usually a very distinct undulating line. The upper border is not so sharply defined; it corresponds to the junction of the lesser wing with the orbital part of the frontal bone. The

**Pathological.**—Diminished density of the lesser wing is sometimes found with intracranial tumours. It may be limited to the centre, or may affect the whole of the lesser wing, so that its density is no longer any greater than that of the adjacent bony orbit, and the sphenoidal fissure can only be discerned with great difficulty. This change may be unilateral or bilateral. It is usually due to pressure from within the cranium. It might be thought that diminished density of the sphenoidal ridge indicates a nearby tumour, but this is only sometimes true, for experience has shown that it may be associated with tumours at a distance. In fact, tumours in almost any part of the brain can produce this appearance, sometimes on one side only, and then either ipsilateral or contralateral to the lesion. Unless interpreted with caution, therefore, diminished density of one lesser wing may be a misleading sign. In one of the early cases of *Vincent's* series the exploration was done on the wrong side owing to the X ray appearance of diminished density on that side. The only safe rule in cases where there is obvious rise of intracranial pressure is to regard diminished density of one lesser wing as of no localising value and merely as radiological indication of raised intracranial pressure. *David* thinks that it may be due to dilatation of the ipsilateral ventricle, and that this accounts for the frequency with which meningioma of one lesser wing of the sphenoid is associated with diminished radiological density of the lesser wing of the opposite side. In *Vincent's* whole series of twenty three meningiomas of the lesser wing, *David* found erosion of the lesser wing on the side of the tumour in five cases, and on the opposite side in three.

Diminished density, due to destruction of the lesser wing of the sphenoid, is sometimes found when the bone is invaded by secondary neoplasm, but it is then often associated with thickening of other parts of the lesser wing from formation of new bone. Diminished density of the lesser wing is also seen, though rarely, as a result of extradural cysts arising from the sphenoidal sinus, and in congenital absence of the superior orbital walls.

Increased density may be a normal phenomenon associated with thickening of the rest of the cranium, and it is not likely to cause confusion in diagnosis when it is bilateral. However, if intracranial pressure becomes raised by a tumour developing within such a skull, there may be thinning of one lesser wing, and the other hypertrophic wing may then suggest the presence of pathological bony thickening. True pathological bony thickening is found in a certain percentage of meningiomas of the lesser wing, in seven of twenty three cases reported by *David*. It may be localised to part of the lesser wing or may be generalised: it then frequently spreads in one of three directions: medially into the anterior clinoid process and sella turcica, or laterally and forwards to the outer wall and roof of the orbit or to the region of pterion (Fig. 57), or backwards into the middle fossa. When the orbit is involved unilateral exophthalmos occurs, often of extreme degree. With widespread hyperostosis of

this nature the underlying meningioma is usually *en plaque*. These appearances may be produced also by metastatic tumour of the lesser wing and adjacent bone.

### Unilateral Exophthalmos

The importance of radiological examination of the orbit and optic canal in cases of unilateral exophthalmos cannot be too strongly stressed for in a considerable number of cases the causal condition arises within the cranium.



FIG. 57.—Hyperostosis of sphenoid and roof of orbit in a case of meningioma *en plaque*. Progressive proptosis of one eyeball for eight years. Woman aged 47. Case No. 1766.

or extends from the orbit into the cranium without giving any clinical sign of intracranial disorder. The following are the most important causes of exophthalmos:

(1) **TUMOUR OF THE ORBIT**—A tumour may arise in the optic nerve and extend backwards through the optic canal in which case the canal can usually be seen radiologically to be enlarged. Most orbital tumours, however, do not produce radiological signs though some meningiomas are calcified.

(2) **MUCOCELE OF FRONTAL OR ETHMOIDAL SINUSES**

(3) **OSTEOMA OF THE ORBITO-ETHMOIDAL REGION**

(4) **INTRACRANIAL TUMOURS** invading the roof of the orbit such as

meningioma and secondary carcinoma, or pressing on the roof of the orbit, causing excessive resorption and thinning, such as cholesteatoma

(5) LESIONS OF THE CAVERNOUS SINUS including arterio venous aneurysm

(6) CONGENITAL DEFICIENCY OF THE ROOF OF THE ORBIT

(7) EXOPHTHALMIC GOITRE and the allied condition of exophthalmic ophthalmoplegia, described by *Russell Brain*

### The Optic Canal

**Normal**—The optic canal runs from the outer aspect of the optic groove to the orbit, and through it pass the optic nerve and the ophthalmic artery. It is 4 to 8 mm in length and is directed downwards and outwards making an angle of 30 degrees with the sagittal plane. The average measurements of the normal canal are  $4.2 \times 4.3$  mm in cross section, and the maximum diameters are  $5.3 \times 5.6$  mm (Fig 17). It is probable that any measurement above 6 mm can be considered as pathological. A measurement of less than 2.8 mm is considered by *Goodwin* to be abnormally small. These absolute measurements are important but they cannot be relied on in diagnosis, since the changes in size of the optic canals produced by disease are usually of smaller range than the normal variations. Consequently, in practice it is better to take radiograms of each optic canal, with identical technique on each side, and to compare the suspected canal with its fellow. For further details the reader is referred to the work of *Goodwin*.

**Pathological**—The optic canal is seen in special projections, already described. When occupied by a tumour it is enlarged. The greatest enlargement is from a glioma of the optic nerve (Fig 58). Enlargement may also be caused by a meningioma or pituitary adenoma extending into the orbit. In pituitary tumours of long standing, where there is great enlargement of the sella and severe loss of sight, the optic canal should always be examined radiologically, for if the tumour extends into the orbit, there is little chance of improving the patient's vision by an operation. Fractures of the anterior fossa sometimes extend back into the optic foramen, producing partial or complete



FIG 58—Enlargement of optic foramen due to glioma of the optic nerve. Boy aged 7 years. Case No 1177

blindness on the affected side. These fractures are very difficult to see in radiograms.

In special projections for the optic canal the sphenoidal fissure is well displayed below and to the outer side of the optic canal. *Jefferson* and *Twining* have pointed out that the medial end of this fissure may be enlarged in intracranial aneurysms and at the same time the bridge of bone between the fissure and the canal becomes thinned.

### The Petrous Bone and Internal Auditory Meatus

The petrous bone is well displayed in stereoscopic antero-posterior radiograms running across the lower half of the orbit or if the tube is tilted in a



FIG. 59.—Fronton of apex of left petrous bone in a case of acoustic neurinoma. Woman, age 15<sup>9</sup>. Case No. 1448.

cranial direction, appearing above the supra-orbital ridge. It is a very dense bone and it does not often show gross changes in intracranial tumours. Occasionally the apex of the petrous bone is destroyed by an acoustic tumour (Fig. 59) or by secondary carcinoma. This last is a not uncommon lesion, the carcinoma spreading from a concealed primary carcinoma of the naso-pharynx and giving rise to symptoms of a tumour of the cerebello-pontine angle. Cyst-like spaces also are found in cholesteatoma of the petrous bone (*Jefferson* and *Smalley*).

Radiological examination of the middle and internal ear is a special study in connection with otology and the internal auditory meatus is the only part of the ear on which the radiologist is asked to report for the purposes of neurological diagnosis. The methods by which the internal auditory meatus can be displayed have already been described. The value

of the information obtained is not great, but will be discussed further when acoustic neurinomas are being considered (see Chapter IV)

By his detailed studies of the petrous bone *Steniers* has been able to display many fractures of the petrous bone that are missed in ordinary radiograms, and he has also shown in a certain number of acoustic tumours not only widening of the internal auditory meatus, but also diminished density of the apex of the petrous bone from pressure of the tumour lying behind it

## CALCIFICATION

### A. Physiological

Calcification occurs as a normal phenomenon in adults in the pineal body, the choroid plexuses of the lateral ventricles, and in certain parts of the dura mater. Bone also may be found in the dura

**The Pineal Body**—Calcium is deposited in the central part of the pineal body as age advances. It has no pathological significance, and probably represents a normal degenerative change in a structure which in foetal life and infancy is histologically active and perhaps exerting some as yet unknown function. The frequency with which calcification is seen radiologically throughout all ages is about 50 per cent. It is seen rarely in the first decade, in about 20 per cent of individuals in the second decade, and thereafter it is much more common.

The pineal body lies in the middle line slightly above and behind the level of the petrous bone. When calcified it is readily seen in lateral pictures, but not so easily in the antero posterior plane, where its shadow is apt to be obscured by the accessory sinuses or by the density of the shadow of the occipital bone. The best method of displaying it in this plane is to use the antero posterior projection with 35 degrees tilt (Fig. 12).

The pineal shadow varies in size from a pin head up to 1 cm. in its longest, antero posterior diameter. It may be a single small circular opacity, or a round or oval cluster of small calcified areas. An area of calcification in the pineal region larger than 1 cm. in its longest diameter suggests the possibility of a pineal tumour, especially in a patient below the age of 20, but no radiological diagnosis of pineal tumour is conclusive without ventriculography.

The pineal shadow is often displaced from its normal position by expanding intracranial lesions, and this fact has been used by *Naffziger* as an indirect method of diagnosing the situation of the lesion. Taking true antero posterior pictures by the method described above, he has found that when intracranial pressure is raised, as shown by the observation of papilloedema and other clinical signs, lateral displacement of the pineal shadow indicates an expanding lesion in or on the opposite cerebral hemisphere. In some cases this may prove a valuable aid in diagnosis (Fig. 60). *Pastine* and *Kinney*, and *Lalja*



have attempted to measure displacement of the pineal body in horizontal and coronal planes. They have been able to show that vertical and horizontal displacements of the pineal shadow do occur in intracranial tumours but their method is not as yet precise enough to be of value in diagnosis since *Dyke* has demonstrated the pineal shadow of certain normal individuals especially those with dolichocephalic skulls may be outside the limits of the normal zone.

**The Choroid Plexus**—Calcification may occur in the choroid plexuses of the lateral ventricles and rarely ossification as a sequel to the degeneration of vascular loops of the plexus and of the connective tissue around



FIG. 60 Displacement of pineal shadow to the left in a case of a very large spongioblastoma multiforme of the right occipital and temporal lobes in a woman aged 53. Case No. 3906.

them. Each vessel is replaced by a tube or ring of calcium and other salts. The process occurs most commonly (or most intensely) at the junction of the body and inferior horn of the lateral ventricle and when the calcified rings are numerous they cast a faint shadow which varies usually from 0.5 cm to 1.5 cm in greatest diameter (Fig. 61). The shadow is seen to be composed of a number of small calcified points; its outline is usually slightly crenated conforming to the outline of the choroid plexus. It is usually bilateral and the two shadows lying 1.2 cm below and 2.3 cm behind the pineal shadow make with it an equilateral triangle when viewed stereoscopically. Only one choroid plexus may throw a shadow and there is a risk that it may be confused with the pineal shadow. The shadow of a calcified choroid plexus is usually larger and fainter than the pineal shadow. It is situated farther back and at a lower level than the pineal shadow and occurs less frequently, being present in about 5 per cent

of individuals of all ages (*Dyle*) Unilateral calcification of the choroid plexus may simulate the calcification of a deep seated glioma but the diagnosis can be settled by ventriculography

Occasionally the whole of the choroid plexus may cast a shadow as in the



FIG. 61—Calcified choroid plexuses. Antero-posterior and lateral projection

case of a girl who had a meningioma of the right cerebello pontine angle. In another case a man with bilateral acoustic neurinomas and multiple meningiomas both lateral choroid plexuses were completely solid and visible in radiograms. Histologically they were composed of very numerous psammoma bodies.



FIG. 62—Regeneration of bone on the dura over the cerebellum nine years after cerebellar decompression in a 15 year old girl for unverified cerebellar tuberculoma. Several silver clips are also seen. Case No. 77

**The Dura Mater**—The dura mater has the power to form bone, as is shown by the new bone that often appears in subtemporal or cerebellar decompressions particularly in children (Fig 62). It is therefore not sur

prising to find bony plaques on different parts of the dura as a normal phenomenon. They are found most commonly on the falx (in nearly 7 per cent of normal individuals according to *Dyle*), and though attempts have been made to vest them with pathological significance, no convincing case has as yet been made. These plaques can occur on any part of the falx and are of varying dimensions. They are best seen in antero-posterior projections, and are often difficult to discern in lateral pictures, on account of their thinness. Sometimes they have a low conical form as seen in antero-posterior pictures, and may be mistaken for calcification in the base of a meningioma of the falx itself a rare phenomenon. There is no radiological method of distinction other than ventriculography. Symmetrical plaques are sometimes seen in the dura on either side of the sagittal sinus and have been called, by *O Sullivan*, *retrobregmatic ossifications*.

Other parts of the dural envelope may become opaque to X rays. Bony or calcified plaques may occasionally be seen in the tentorium cerebelli. The petro-clinoid ligaments, running from the apex of the petrous bone to the corresponding posterior clinoid process, are sometimes ossified (Fig. 44), and are then seen in lateral radiograms as linear shadows behind the dorsum sellæ.

## B. Pathological

Thin plaques of calcified material may occasionally be seen, especially in children, scattered over the dura of the convexity of the cerebral hemisphere, or over the dura of the cerebellum, as a result of long-continued intracranial pressure. It is probable that they represent the final result of areas of hyaline degeneration so commonly seen as yellow flecks in the dura in cases of chronically raised intracranial pressure.

**Dural and Subdural Calcification**—Fragments of ectopic bone are occasionally found lying loose on the surface of the brain, usually on the convexity near the sagittal sinus. In the cases we have seen they were associated with epilepsy due to birth injury.

A few cases of very chronic subdural hæmatoma have been reported in which the whole of the clot was calcified. Calcification may occur in chronic otitis in the dura over the tegmen tympani.

**Brain Calculi**—Multiple or single patches of calcification may occur in the brain substance, usually in the form of small circular or triangular areas of uniform density with sharp margins. They are usually associated with epilepsy and mental deficiency, and sometimes with hemiplegia. Sometimes there is history of injury, when the calculi are probably the result of old intracerebral hæmorrhage, often at birth or in infancy. Often, however, the condition is familial, and the calcium is deposited symmetrically in the basal ganglia, dentate nuclei, and is widely spread throughout the white matter. *Dandy* has observed large, bilaterally symmetrical, areas of calcification in the walls of the anterior horn of each lateral ventricle. The calcium appears to be

deposited in the first instance in the walls of the capillaries and then spreads to the perivascular spaces and adjacent tissue, and one area fuses with another. *Rio Hortega* has studied a case in which there was widespread deposit of calcium within the nerve cells of the cortex.

**Neoplastic Calcification**—Calcification occurs in almost any variety of intracranial tumour. It is most common in the *glioma group*, particularly in supratentorial astrocytoma and oligodendroglioma, but also in ependymoma and in the more malignant glioblastoma multiforme. The calcification may be very dense or scanty, diffusely scattered through the tumour, or limited



FIG. 63.—Calcified astrocytoma in right basal ganglia. Woman aged 23. Case No. 2129.

or one small part of it. It scarcely ever indicates the true size of the tumour. When it is in the form of a spray of fine lines or dots it usually indicates an astrocytoma (Fig. 63). A broad line of calcification of varying width up to 1 cm., extending in a rather tortuous course for distances up to several centimetres, usually signifies an oligodendroglioma (Fig. 143). Apart from these two varieties there is nothing in the appearance of the calcification that gives a clue to the type of glioma. The presence of extensive calcification in a tumour suggests that the tumour is of slow growth and one in which the prognosis after surgical removal should be favourable; but this is not invariably true. There is a growing volume of evidence in favour of the view

that some gliomas tend to become more primitive in type, and more malignant, as their growth proceeds. A glioma may show well differentiated and orderly growth in one place, and undifferentiated and unmistakably malignant type of growth in another.

In a number of gliomas the area of calcification is extremely small, sometimes no more than a few flecks that can only be found by the most careful stereoscopic study of radiograms taken with soft penetration. It is curious that calcification is scarcely ever seen in subtentorial tumours, though it has been reported by *Masson* in a cerebellar medulloblastoma. Some of the most extensive calcifications are found in the cerebral gliomas of young children.



FIG. 64—Intracranial teratoma in a male aged 24 years showing numerous teeth. There is also enlargement of the head. (Dr R. A. C. Rigby's case.)

The situation of the shadows within or above and behind the sella, makes the diagnosis of the pathology of the shadow practically certain, and the main difficulty in radiological diagnosis is the demonstration of the calcification when it is slight. Sometimes the shadows are so small and faint that they are only detected in underexposed films; at other times so dense and extensive as to indicate clearly, not only the pathology of the tumour, but also the great surgical risks entailed in its removal.

*Tuberculoma* (Fig. 147) tends to become calcified in its chronic stage, and then is frequently multiple. As *Purdon Martin* has shown, the shadow may be spherical or lobulated, it varies in density, being usually greatest at its edges, which are sharply defined and crenated.

*Intracranial osteomas* give a very sharply defined, homogeneous shadow

*Meningiomas* are sometimes calcified, owing to the formation of many psammoma bodies in their substance, and the tumour is then visible through the whole of its extent as a homogeneous shadow, usually of little density, with a circular or lobulated outline (Fig. 139). At other times the tumour may be calcified in one part only (Fig. 140) and then most often at and near its point of attachment to the dura or else where at the periphery. Ossification may also occur in a meningioma (Fig. 1).

*Epidermoid tumours* (cranio-pharyngiomas) are commonly calcified and rarely ossified.

denser than in other tumours. They are usually attached to the base of the skull.

*Angiomatous formations* when calcified show a characteristic shadow in a background of stippled shadow there are double parallel lines of greater density which from their size and appearance suggest calcification in the walls of blood vessels though in fact the calcium is deposited not in the vessel

A



Fig. 151.—Transverse angioma of right frontal lobe. Head collapsed on with fracture through table and injury to right optic nerve and both olfactory tracts. Eleven weeks later cerebral hemorrhage and intermittent left hemiparesis. The area shown in the radiogram entirely within the frontal lobe. The double parallel project on into the angioma (A) corresponds to sulci. Case No. 39.

cells but in the brain cortex (Fig. 151). In the arterio-venous type of angiomatous malformation a small fleck of calcification may be seen which resembles the calcification of some gliomas.

*Other Tumours*—Among common tumours that rarely cast shadows must be mentioned pituitary adenoma and metastatic tumour. Teratoma a rare tumour may cast a dense shadow even showing the formation of teeth (Fig. 64). In cysticercosis the intracerebral cysts occasionally become calci-

fied, like cysticerci elsewhere in the body, and show as multiple small faintly calcified nodules (Fig 148)

### Cerebral Aneurysm

Aneurysm of the internal carotid or main cerebral arteries sometimes becomes calcified in its wall and shows in radiograms as a circle or part

of a circle (Fig 150). Linear streaks of calcification are rarely seen along the course of the main intracranial arteries in severe atheroma.

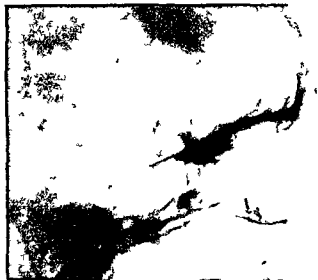


FIG 67. Supra-ellalar aérocele found after head injury in a woman aged 34 who showed bilateral temporal hemianopia and failure of vision. The symptoms cleared up completely with absorption of the air and did not recur in the ensuing five years. The sella turcica shows gross destruction. Case No 1080.

the tumour in its expansion destroys the overlying skull and deforms the orbit (Fig 162). Air has also been found in cystic cavities at the base of the brain such as those of epidermoid cysts (Fig 66). In an abscess of the brain gas is often formed but only on rare occasions is it in sufficient quantity to be demonstrable radiologically.

### AEROCELE

Air may make its way into the cranium in case of fracture of the anterior and middle fossæ where there is usually also cerebrospinal rhinorrhœa. The air may be subdural (Fig 83) or intracerebral (Fig 65) and may eventually find its way into the ventricles producing a spontaneous ventriculogram. Similar appearances are sometimes seen in cases of osteoma of the accessory sinuses of the nose where

### BIBLIOGRAPHY

- BOCAGE A F M French Patent 1922 No 536464  
 BRAIN W R Exophthalmic Ophthalmoplegia *Trans Ophthalmolog Soc* 1931 LVII 107  
 CAMP J D Intracranial Calcification and its Roentgenologic Significance *Ann Journ Roentgenol* 1930 XXIII 615 The Roentgenologic Manifestations of Intracranial Disease *Radiology* 1939 XIII 484

- CAMP, J. D., and GIANTURCO, C. A., "A Simplified Technique for Roentgenographic Examination of the Optic Canals," *Amer Journ Roentgenol*, 1933, XXIX, 547.
- CAPR, G. L., "Roentgen Ray Findings in the Skull in Cases of Brain Tumours," *Amer Journ Roentgenol*, 1917, IV, 405
- CHAUMET, G., "Traité de Radiodiagnostic," 1933, Paris, Vigot
- DALE, T., "Intracranial Calcification," *Acta Radiol*, 1934, XV, 628
- DAVID, M., "Les méningiomes de la petite aile du sphénoïde," 1933, Paris, Vigot
- DAVID, M., and STUHL, L., "Les Méningiomes de la petite aile du sphénoïde étude radiologique," *Journ de Rad et d'Elect*, 1933, XVII, 193
- DAVIS, L., "Intracranial Tumours," *Ann of Roentgenol*, 1933, XIV, New York, Hoeber
- DYKE, C., "Indirect Signs of Brain Tumour," *Amer Journ Roentgenol*, 1930, XXIII, 598
- ELLSBERG, C. A., and SCHWARTZ, C. W., "Increased Cranial Vascularity in its Relation to Intracranial Disease," *Arch Neurol and Psychiat*, 1924, XI, 293
- FRAY, W. W., "Roentgenologic Study of Orientation of the Pineal Body," *Arch Neurol and Psychiat*, 1937, XXXVIII, 1199
- EYELIN, H. R., and PENFIELD, W., "Cerebral Calcification Epilepsy," *Arch Neurol and Psychiat*, 1929, XXI, 1020
- GALWIN, H. A., "The Precise Roentgenography and Measurement of the Optic Canal," *Amer Journ Roentgenol*, 1925, XIII, 480, "One Thousand Optic Canals," *Journ Amer Med Assoc*, 1927, LXXXIX, 1745
- HASHE, R., "Atlas typischer Röntgenbilder von normalen Menschen," Lehmanns Medizinische Atlanten, 1912, München, Lehmann
- HEUSCHEN, F., "Die Akustikustumoren eine neue Gruppe radiographisch darstellbarer Hirntumoren," *Fortschr a a Geb d Röntgenstrahl*, 1911, XVIII, 207
- KEUER, G., and DANDY, W. E., "Roentgenography in the Localisation of Brain Tumour," *Bull Johns Hopkins Hosp*, 1916, XXVII, 309
- FORTEGA, P. DEL RIO, Personal Communication
- EFFERSON, G., "Radiography of the Optic Canals," *Proc Roy Soc Med*, 1936, XXI, 1169
- EFFERSON, G., and SMALLEY, A. A., "Progressive Facial Palsy produced by Intratemporal Epidermoids," *Journ Laryngol and Otol*, 1938, LIII, 417
- EFFERSON, G. and STEWART, D., "On the Veins of the Diploe," *Brit Journ Surg*, 1928, 29, XVI, 70
- EPE M. H., "The Reaction of the Bones of the Skull to Intracranial Lesions," *Brit Journ Radiol*, 1938, XI, 146
- FRAYENBUHL, M., "Hilfsmethoden der Diagnostik raumbeschränkender, intrakranieller Erkrankungen," *Schweiz med Woch*, 1937, LXVII, 89
- FRAY W. L. T., "Dilatation of Diploe Veins and other Anatomical Variations in the Skull," *Amer Journ Roentgenol*, 1925, XII, 536
- ILJA, B., "Localisation of Calcified Pineal Bodies," *Acta Radiol*, 1934, XI, 659
- JANDBLOM, K., "Roentgenographic Study of the Vascular Channels of the Skull," *Acta Radiol*, 1936, Suppl. XXX
- JASHOLM, F., "Apparatus and Technique for Roentgen Examination of the Skull," *Acta Radiol*, 1931, Suppl. XII
- JARTIN, J. P., "Calcified Intracranial Tuberculomata," *Brit Journ Radiol*, 1937, X, 5
- JARTIN, P., and CRUICKSHANK, H., "Primary Gliomas of the Chiasm and Optic Nerves in their Intracranial Portion," *Arch Ophthalmol*, 1923, LII, 209
- JASSON, C. B., "The Occurrence of Calcification in Gliomas," *Bull Neurol Inst of New York*, 1931, I, 314
- JAYER, E. G., "Otologische Röntgendiagnostik," 1930, Berlin
- JERRITT, H. H., and FREMONT SMITH, F., "The Cerebro-spinal Fluid," 1937, Philadelphia
- LAFTZIGER, H. C. A., "A Method for the Localisation of Brain Tumours, the Pineal Shift," *Surg Gynec and Obstet*, 1925, XL, 481
- SULLIVAN, J., "Some Rarer Intracranial Calcifications and Ossifications," *Brit Journ Radiol*, 1925, XXX, 295



- SCHULLER A    Roentgen Diagnosis of Diseases of the Head    1918 St Louis C V Mosby  
 Roentgendiagnose der Akustikustumoren    *Ergebn d med Strahlenfor* 1918 III 89
- SOSMAN M C    The Reliability of Roentgenographic Signs of Intracranial Tumour  
*Amer Journ Roentgenol* 1936 XXXVI 737    Eighteenth Annual Report of Pe  
 Bent Brigham Hospital for 1931 1932 Boston
- STENVERS H W    Roentgenology of the Os Petrosum    *Arch Radiol and Electr*  
 1917 XXII 97    \* Roentgenology of the Os Petrosum    *Acta Otolaryngol* 19  
 III 266
- TAYLOR JULIAN    Invasion of the Skull by Dural Tumours    *Brit Journ Surg* 19  
 XVI 6
- TOWNE E B    Erosion of the Petrous Bone by Acoustic Nerve Tumors    *Ar*  
*Otolaryngol* 1926 IV 515
- TWINING E W    Tomography by Means of a Simple Attachment to the Potter Bus  
 Coucl    *Brit Journ Radiol* 1937 X 332    Posture in Radiography of the Skull  
*Proc Roy Soc Med* 1933 34 XXVII 591    The Value of Radiology in Neu  
 surgery    *Proc Roy Soc Med* 1936 XXI 1155
- VASTINE J H and KINNEY K K    Pineal Shadow as an Aid in the Localisation  
 Brain Tumours    *Amer Journ Roentgenol* 192 XXII 320
- ZIEDESS DES PLANTES B G    New Method of Differentiation in Radiography (Pla  
 graphy)    *Acta Radiol* 1939 XIII 182

## CHAPTER III

### VENTRICULOGRAPHY AND ENCEPHALOGRAPHY

CONTRAST MEDIA were first used in radiographic study of the cerebrospinal fluid pathways in 1918 when *Dandy* injected air into the ventricles in cases of infantile hydrocephalus. The method was soon found to be of great value in localising intracranial tumours that gave no certain indications of their site by clinical and ordinary radiological methods. The air was introduced by boring a hole in the skull and tapping the ventricle, but later *Dandy* found

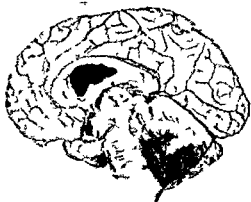


FIG. 67.—Cerebellar medulloblastoma. Note the white plaques of metastatic tumour on the medial aspect of the frontal lobe. Case No. 35a.

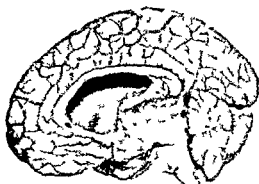


FIG. 68.—Glioma of the posterior half of the third ventricle and adjacent thalamus producing gross dilatation of the lateral ventricles. Case No. 187.

that satisfactory pictures could also be obtained in certain cases when it was injected by lumbar puncture with the patient sitting up. By this route not only the ventricles but also the subarachnoid spaces were filled. This method is called *encephalography* as distinct from *ventriculography*, in which the air is introduced directly into the ventricles.

The value of contrast media in the ventricles depends upon the fact that practically every expanding and every contracting lesion of the brain produces some deformity of the ventricular system. Tumours of the posterior fossa sooner or later block the fourth ventricle and produce dilatation of the third and lateral ventricles (Fig. 67). Tumours in the third ventricle and its neighbourhood also produce symmetrical dilatation of the lateral ventricles, but they obliterate part or the whole of the third ventricle (Fig. 68). A

tumour in one cerebral hemisphere displaces the lateral and third ventricles to the opposite side—the lateral ventricle on the side of the lesion tends to become collapsed while that on the opposite side is usually dilated (Fig 69). This displacement is not due to the bulk of the tumour alone but also to oedema of the surrounding white matter for it will occur even when the

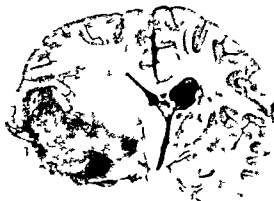


FIG 69 Ventricular displacement produced by a spongioblastoma of the temporal lobe. Case No 119



FIG 70—Ventricular displacement produced by a meningioma on the surface of the frontal lobe. There is great oedema of the white matter beneath the tumour. Case No 93

tumour is quite small and at a considerable distance from the ventricles (Fig 70). Similar alterations of the ventricular system are produced by abscess, massive hæmorrhage and other coarse lesions of an expanding character.

Scarring of the brain following severe injuries or degenerative lesions is associated with dilatation of the adjacent part of the ventricular system and often of the overlying subarachnoid space. In these cases such lateral displacement of the ventricles as occurs is towards the side of the lesion.

## THE ANATOMY OF THE CEREBROSPINAL FLUID PATHWAYS

Most if not all of the cerebro-spinal fluid is formed by the choroid plexuses of the ventricles. Its course is shown in Fig 71. From each lateral ventricle fluid passes through the corresponding foramen of Monro into the third ventricle then through the narrow aqueduct of Sylvius into the fourth ventricle. It leaves the fourth ventricle by one posterior opening, the foramen of Magendie, and two antero-lateral openings, the foramina of Luschka, which lie one on each side of the medulla oblongata. The fluid then enters the subarachnoid space and ultimately finds its way to the surface of the cerebral hemispheres where it is absorbed into the dural blood sinuses through the arachnoid villi.

clusters of endothelial cells in the arachnoid membrane, which are embedded in the superior longitudinal and other venous sinuses. When hypertrophied with advance of years, the villi are called Pacchionian granulations. Some cerebrospinal fluid may also be absorbed by the capillaries within the brain itself, or by the choroid plexuses, but this is probably only a small fraction.

In normal adults the amount of cerebrospinal fluid in the ventricular and subarachnoid spaces varies between 70 and 180 cc. The normal lateral ventricle has a capacity of 8-15 cc. but when dilated in severe hydrocephalus from tumour or any other cause it may contain over 100 cc of fluid. The normal pressure of this fluid in the ventricle or in the lumbar

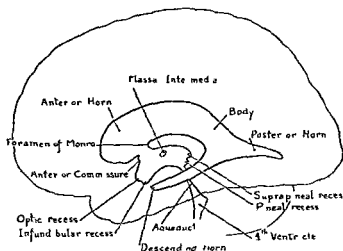


FIG. 71.—Diagram of ventricular system.

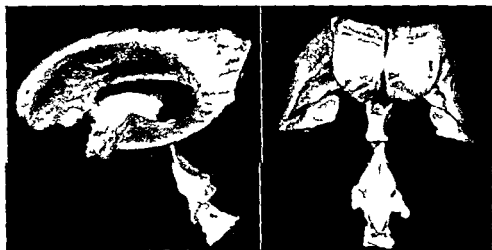


FIG. 72.—Model of the ventricular system. Lateral and antero-posterior projections (After Petrus).

subarachnoid space, with the patient relaxed in the horizontal position, varies between 70 and 180 mm. of water. Increase of intracranial venous pressure, as by coughing or compression of the internal jugular veins in the

neck, produces a prompt rise of cerebrospinal fluid pressure in the ventricles and in the lumbar subarachnoid space

The normal rate of formation of cerebrospinal fluid in man has been estimated at about 400 c.c. a day, but there must be considerable variations depending on posture, exertion and other conditions which influence venous pressure and on the ingestion of fluids and salts which influence the osmotic

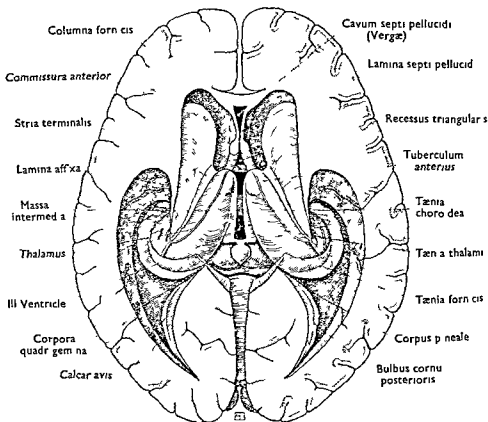


FIG. 73.—Horizontal section through the brain (Villiger) (From Lissolm)

pressure of the blood. The rate is altered in a variety of pathological conditions. In intracranial tumour in certain conditions the rate of formation is evidently increased to many times normal. Injection of air into the ventricle probably increases the rate of formation.

The anatomy of the ventricles and their relationship to one another can best be studied by means of a model (Fig. 72).

The Lateral Ventricle (Fig. 73) consists of a body, anterior, posterior, and descending horns.

The ANTERIOR HORN is that part of the ventricle which lies anterior to the

foramen of Monro. It is roughly triangular in section, and as it passes forwards over the head of the caudate nucleus it gradually curves downwards and laterally. The anterior horns are separated from one another posteriorly only by the thin septum lucidum, and anteriorly by a considerable amount of brain tissue. The roof and anterior wall of the anterior horn are formed by the corpus callosum; in the lateral wall is the caudate nucleus. The septum lucidum, which constitutes the medial wall, is composed of two thin layers within which lies a small space, the *cavum septum pellucidum*, or *cavum Vergæ* (Fig. 74).

THE BODY OF THE LATERAL VENTRICLE extends backwards from the foramen of Monro to the trigone, which is the meeting-place of the body, posterior, and descending horn. It is roughly triangular in section, its roof being formed by the corpus callosum, its medial wall by the septum lucidum, and its floor from without inwards by the caudate nucleus, the choroid plexus, and the upper surface of the fornix. Beneath the choroid plexus and the fornix at this level lies the optic thalamus.



Fig. 74 —Antero posterior projection, showing a large *cavum pellucidum* (Vergæ)

THE POSTERIOR HORN passes from the posterior end of the body of the ventricle into the occipital lobe. Its length is very variable, up to 3 cm.; not infrequently it is totally absent on one or both sides. On section it is roughly circular. Its medial wall presents two projections, the upper the bulb of the posterior horn, the lower the calcar avis, which corresponds to the anterior part of the calcarine fissure.

THE DESCENDING HORN (Fig. 71) is a continuation of the lateral ventricle into the temporal lobe. It arises from the posterior part of the body of the ventricle and passes forwards, downwards, and laterally around the optic thalamus and lenticular nucleus. The anterior tip of the horn curves medially and sometimes downwards, ending 2 to 3 cm. behind the extremity of the temporal pole. The medial part of the floor of the descending horn presents throughout the whole of its course a prominent elevation, the hippocampus, which is partly covered by choroid plexus. This elevation appears at times to prevent complete filling of the descending horn with air, so that in ventriculograms the horn looks extremely narrow.

The Third Ventricle (Fig. 75), into which the lateral ventricles open through

the foramina of Monro is a narrow vertical cleft, situated in the middle line between the two optic thalami. It measures 2.5–3 cm in height and is slightly longer in its antero-posterior axis. At its anterior-inferior extremity

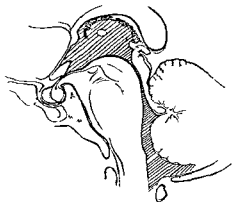


FIG. 70. Diagram of third and fourth ventricle and aqueduct of Sylvius in sagittal section.

it presents a funnel-shaped extension into the stalk of the pituitary body (recessus infundibuli), and another recess above the optic chiasm (recessus opticus). In its centre the third ventricle is crossed by the middle commissure (massa intermedia), a band of grey matter of variable size joining the optic thalami. At its posterior-inferior end the third ventricle opens into the aqueduct of Sylvius. Above this opening is a small recess into the stalk of the pineal body (recessus pinealis). Anteriorly, the floor of the ventricle is largely formed by the tuber cinereum and contains the hypothalamic

nuclei while posteriorly it is formed by the corpora mamillaria and cerebral peduncles. The anterior wall extends from the optic chiasm to the anterior commissure which lies immediately in front of the foramina of Monro and is formed by a thin layer of grey matter (lamina cinerea). On the roof is the velum interpositum, an invaginated fold of the pia mater containing the two choroid plexuses of the third ventricle, and above that lie the fornix and the corpus callosum.

The Aqueduct of Sylvius is a narrow channel passing through the midbrain to connect the third with the fourth ventricle. It passes downwards and slightly backwards and is 1.5–2 cm long and about 0.2 cm in diameter. Above and behind it is the quadrigeminal plate, below and in front the tegmentum of the crura cerebri.

The Fourth Ventricle is triangular in lateral projections and rhomboidal in form in antero-posterior projections. In front of it lie the pons and medulla oblongata, the posterior surfaces of which form the floor of the fourth ventricle. Behind it lies the cerebellum. At its lower end posteriorly in the middle line is the foramen of Magendie, leading into the cisterna magna, while laterally the lateral recesses of the ventricle lead through the foramina of Luschka into the cisterns of the cerebello-pontine angles.

The Subarachnoid Space communicates with the ventricular system by means of the foramen of Magendie and the foramina of Luschka. At the base of the brain and at the junction of the mid and hind brain at the opening in the tentorium cerebelli it forms a series of cisterns of considerable extent of which the largest is the cisterna magna lying over the back part of

the cerebellar tonsils and medulla oblongata. On the base of the brain extending from behind forwards are the *cisternæ pontis*, *interpeduncularis*, and *chiasmatis*. There are other cisterns, some named and others unnamed. They are frequently outlined in encephalograms. In passing from the cisterns of the posterior fossa the cerebrospinal fluid must go through the opening of the tentorium cerebelli. Here the subarachnoid space is narrow and it is therefore not surprising that blockage commonly occurs at this site, producing external (or communicating) hydrocephalus.

Where the arachnoid passes over the summits of cerebral convolutions it is so closely bound to the subjacent pia mater that the subarachnoid space is virtually obliterated, but in the intervals between the convolutions in the fissures or sulci the subarachnoid space is wide enough to be seen in encephalograms as lines of air that correspond in size and direction to the fissures of the brain.

### NORMAL RADIOLOGICAL APPEARANCES

In normal people the ventricular system shows such considerable variations in size that no precise measurements have been established. The distinction between ventricles that show a small degree of hydrocephalus and ventricles



FIG. 76.—Ventriculogram and diagram of normal lateral projection. The numbers are explained in the text.



that are within the upper limits of normal often cannot be made from the study of ventriculograms, and appearances can only be interpreted in the light of the history and physical signs. Variations in shape and extent occur in the ventricular horns from one normal subject to another, and, in the same subject, from side to side. The anterior horn may show a small indentation at its postero inferior limit, the posterior horn is not infrequently absent on one or both sides and may show many other variations, and the descending horn

varies greatly in the length and direction of its anterior extremity. In the third ventricle the middle commissure is sometimes so large as to simulate a tumour.

The lateral projection offers no difficulties of interpretation (Fig 76). In the antero posterior and postero anterior projections shadows may be superimposed on one another and they require some analysis. This can best be done with a ventricular model, after the methods of *Locke*, and *Torkildsen*. Fig 77 shows the normal antero posterior view and a diagram interpreting the shadows in the light of a ventricular model. Shadow 1 is cast by the anterior horn, shadow 2 by the junction of the anterior horn with the body of the

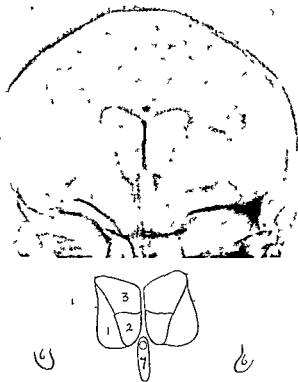


FIG. 77.—Ventriculogram and diagram of normal antero posterior projection. The numbers are explained in the text.

ventricle, shadow 3 by the main part of the body of the ventricle, shadow 4 by the posterior part of the body of the ventricle, shadow 5 (not often seen in this projection) by the posterior horn, shadow 6 by the descending horn, shadow 7 by the third ventricle, and shadow 8 by the fourth ventricle. In the postero anterior projection (Fig 78) the shadows are again numbered according to the description given above.

In the antero posterior projection the third ventricle shows as a vertical slit immediately below and slightly overlapped by the septum lucidum. In the postero anterior projection the third ventricle presents a much smaller

shadow, because, as a rule, in the face-down position only its posterior part is occupied by air, and this part has a smaller vertical diameter than the anterior part. The projections necessary to get satisfactory pictures of the third ventricle will be referred to under Technique. The lateral view of the third ventricle is shown in Fig. 76. The shadow is, of course, fainter than those of the lateral ventricles because the third ventricle is so narrow. Interpretation of the shadow presents no difficulties if the ventricle can be well filled by suitable manipulation of the head, though the shadows of the descending horns are often superimposed on that of the lower part of the third ventricle.

The aqueduct of Sylvius and the fourth ventricle are shown in lateral projection in Fig. 76, and in the occipito-frontal (half-axial) position in Fig. 79, this being the best sagittal projection for their display. In the lateral projection the aqueduct shadow is sometimes obscured by the shadows of mastoid cells, and it is crossed by the shadow of the descending horn of the lateral ventricle. The shadow of the fourth ventricle is often partially covered by the shadows of mastoid cells, and its anterior part may be obscured by the dense shadows of the petrous bones. Fig. 80 shows some of the basal



FIG. 78.—Ventriculogram and diagram of normal postero anterior projection. The numbers are explained in the text.



FIG. 79.—Normal occipito-frontal ventriculogram.



FIG. 80 Lateral encephalogram in the face up position showing air in sulci in the anterior horns of the lateral ventricles and in the cisternae ciasmatica and interpeduncularis. Case of gloma of third ventricle. Case No. 3149.

cisterns and sulci. Detail of the normal appearance of these can be obtained from the monograph of Davidoff and Dyle.

During its injection air sometimes passes into the subdural space. Fig. 81 shows subdural air under the tentorium cerebelli and Fig. 82 shows it alongside the falx. In encephalography this is evidently due to rupture of the arachnoid membrane since normally there is no connection between the subarachnoid and subdural spaces. In ventriculography the air gains entry to the subdural space through the hole that is necessarily made in the dura for

the passage of the brain needle and air is most likely to enter in this way in those people whose intracranial pressure is low. Fig. 83 shows air in the subdural space in lateral projection.

### TECHNIQUE

The technique described in this section is the one we employ when using air and any modifications necessary with other contrast media will be mentioned later. The first point to be settled in each individual case is whether the air should be injected into the ventricles (ventriculography) or into the lumbar subarachnoid space (encephalography). Though more simple to perform encephalography is much the more dangerous method in cases of raised intracranial pressure the only safe rule is to reserve en-



FIG. 81.—Postero-anterior projection, showing air beneath the tentorium cerebelli and in normal lateral ventricles.

phalography for those cases which show focal signs or symptoms with any evidence of rise of intracranial pressure, and to perform ventriculography in cases where there is papilloedema drowsiness, intense headache or vomiting. The indication and the decision as to the route employed, should be made by the clinician.

### TECHNIQUE OF VENTRICULOGRAPHY

**The Injection of Air**—The ventricles should be tapped at their best points in order to inject the maximum quantity of air. Some surgeons tap the posterior horns with the patient in the reclining or sitting



FIG. 82.—Postero-anterior ventriculogram showing air in the subdural space alongside the falx and tentorium. In this case there was a glioma of the corpus callosum. Case No. 1874.



FIG. 83.—Subdural air in the frontal region. A lateral section in the face up position. The posterior limit of subdural air is virtually a straight line indicating a fluid level. From a case of epilepsy with considerable amounts of yellow fluid in the subdural space. The picture shows also air in the anterior horns of the lateral ventricles and in the external interpeduncular and chiasmatic cisterns. Case No. 295.

position others tap through the parietal lobes with the patient supine and a few tap the anterior horns with the patient prone. Ventriculography is not a painful procedure and can be done without preliminary sedatives by injecting local anæsthetic into the area of skin to be incised. Burring of the bone, incision of the dura and passage of the needle into the brain do not produce pain. When the ventricle is entered the approximate intraventricular pressure is measured by allowing the cerebrospinal fluid to escape into a manometer. Then the fluid is collected and air, filtered into a syringe through sterile gauze is gently injected in amounts up to 5 cc. at a

time It is useless and harmful to inject air under great pressure for it on forces its way out of the ventricle through the needle or along the needle track Good filling of the ventricle is obtained by taking time over the whole procedure by manipulating the head gently from side to side in the intervals between the injections of air The smaller the ventricle the greater the care and patience necessary At the end of the injection the incisions in the scalp are stitched up in layers and the patient is then ready for radiographic examination

Unless there is some particular contraindication it is generally advisable to tap and inject both lateral ventricles at the same session In cases of dilatation of both lateral ventricles the fact that air injected into one side only does not cross to the opposite side argues strongly for an obstruction at the foramen of Monro by a tumour in the third ventricle but the spread of air to the other lateral ventricle in such cases does not necessarily mean that the foramina of Monro are patent for in hydrocephalus from any cause it is not uncommon to find holes in the stretched septum lucidum through which air can pass freely from one lateral ventricle to the other even though the third ventricle is filled by tumour

In tumour of a cerebral hemisphere the ventricle on the side of the lesion is often so small that air cannot be injected into it In such cases nevertheless it is still advantageous to tap both ventricles for though on the side of the lesion the needle may not find the ventricle it often enters a cyst or meets the resistance of a tumour and thus gives even more direct information of the situation of the tumour and of its character than can be gained by ventriculography

**The Radiographic Projections** —The arrangement of the ventricles and the narrow intercommunications their planes in relation to each other and to the surface of the skull are very complex and it is therefore useful for the purpose of designing projections to have a model of the ventricular system which can be manipulated in relation to the skull Different clinical problems will require a differing set of projections which can only be planned by the radiologist after consultation with the clinician Throughout the examination it must be remembered that lowering the head below the horizontal will allow the air to escape from the ventricular system In every case the first projection should be developed immediately, in order to see how much air the ventricles contain and to obtain a preliminary idea of the radiographic appearances We use an antero posterior projection for this purpose *Lysholm* takes a preliminary film with the tube at the side of the patient and the skull in the antero posterior position (Fig 80), if the depth of air in the frontal horns in this position is less than 2 cm he considers it useless to proceed Radiological examination after air injection should aim as far as possible at display of the whole ventricular system Since it is rare to obtain complete filling of the ventricles with air, it is necessary to manipulate the head, and thus

to carry the air from one part of the ventricular system to the other between the various projections. These can be worked out by the radiologist with the aid of a model as described. They are shown in Fig 84.

Theoretically the Bucky diaphragm should be above the head with the tube under the couch for in this way the air containing space would be nearest to the film. Mechanically this arrangement is difficult and modern technique has largely eliminated the distortion resulting from separation of film and object by increasing film target distance and by the fine focus tube.

The antero posterior projection without tilt of the tube displays the

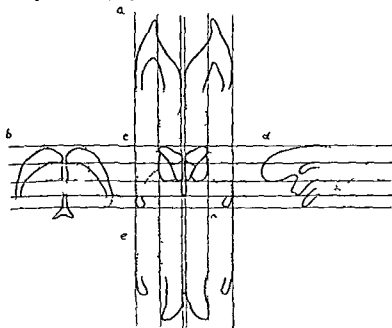


FIG. 84.—Scheme for comparison of ventriculographic appearances in different projections. In each projection the parts of the ventricular system marked with a continuous line are the parts usually seen. *a* Occipito-frontal projection. *b* Postero-anterior projection. *c* Antero-posterior projection. *d* Lateral projection. *e* Oblique antero-posterior projection.

anterior horns, septum lucidum and ventricular bodies, the anterior parts of the descending horns and the anterior part of the body of the third ventricle (Fig 84c). The antero posterior projection with 30 degrees tilt (half axial fronto-occipital projection) shows further details of the anterior horns and bodies. This projection is very important for precise localisation of a tumour in the anterior half of the cerebral hemispheres.

The postero anterior projection with 10 degrees tilt shows the posterior horns, the posterior parts of the bodies and of the descending horns, the posterior part of the third ventricle and occasionally the aqueduct of Sylvius (Fig 84b). The postero anterior projection with 30 degrees tilt (occipito frontal half-axial projection) displays further detail of the posterior horns and trigone (Fig 84a).

and also shows the third ventricle, the aqueduct of Sylvius, and occasionally the fourth ventricle, this projection is very important for precise localisation of a tumour in the posterior part of the cerebral hemispheres

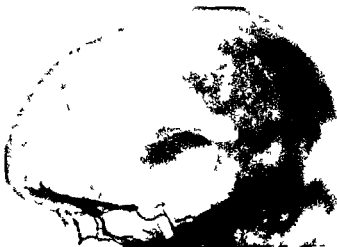


FIG 85—Normal lateral encephalogram with the head erect

appropriate posture for some minutes before the exposure is made in order to allow as much air as possible to get into the uppermost ventricle. The lateral projection usually shows in addition to the uppermost lateral ventricle part of the lower lateral ventricle and part of the third ventricle (Figs 76 and 84d). With good filling, it may show the whole of the third and fourth ventricles and the aqueduct of Sylvius. We prefer to view this projection stereoscopically.

Lateral projections should also be taken with the head in different positions. With the patient lying face upwards a lateral projection shows the anterior and descending horns, the anterior part of the third ventricle (Fig 84d) and also especially in encephalography for traumatic epilepsy and other non expanding lesions air in the subarachnoid or subdural space over the frontal pole (Fig 80). With the patient lying face downwards a lateral projection shows the posterior horns, the posterior parts of the bodies and occasionally the posterior part of the third ventricle, the aqueduct of Sylvius and the fourth ventricle, it also shows any air that may be in the subarachnoid and subdural spaces over the

The lateral projection is designed to show the air in the uppermost lateral ventricle, and if filling is incomplete an apparent defect may be created in the region of the body of the ventricle as this lies at a lower level than the rest of the ventricle when the head is in the lateral position. The patient should thus be allowed to rest in the



FIG 86—Position for lateral projection to show aqueduct and fourth ventricle (After Treuning)

occipital poles With the patient's head erect a lateral projection shows the upper contour of the bodies of the ventricles (Fig 85)

The third ventricle can sometimes be seen well in the ordinary lateral projection but more often the head must be specially placed to display it and it must be studied in two parts For the anterior part of the third ventricle the patient lies on his back and his head is lowered and extended so that the chin points to the ceiling, a lateral view of the skull in this position is then taken (Figs 80 and 84d) The posterior part of the third ventricle the aqueduct of Sylvius and the fourth ventricle are difficult to display unless there is complete filling and also some obstruction to the escape of air into the subarachnoid space They sometimes show well in lateral projections without special manipulation but usually it is necessary to make a lateral projection in the face down position using the following technique the patient is turned on his face but keeps his head extended until a few seconds before the exposure when his head is flexed on his chest the chin being at the same time kept forwards Without such precautions air may escape from the fourth ventricle before the picture is taken In the sagittal plane the projection employed for the fourth ventricle is the postero anterior with 30° tilt The posterior part of the third ventricle the aqueduct and fourth ventricle may also be shown by a lateral projection in the manner described by *Turning* (Fig 86) In this projection the patient lies in the lateral position with his lower shoulder on the couch top His buttocks are raised and his head is tilted towards the floor until the sagittal plane of the skull makes an angle of 45 degrees with the horizontal The central ray is directed through a point 3 cm above the external auditory meatus and the film is placed at right angles to the central ray In order to prevent the air from escaping into the cisterna magna the head should be kept raised until all preparations are complete The picture should be taken immediately after the head has been lowered on to the cassette

The following summary gives the projections in the order most commonly used by us

Patient supine	{	1 Antero posterior without tilt
		2 Lateral in face up position
		3 Antero posterior or fronto occipital (tube 30 degrees headwards)
		4 One lateral (stereoscopic)
Patient prone	{	5 The other lateral (stereoscopic)
		6 Postero anterior with 10 degrees tilt
		7 Postero anterior or occipito frontal (tube 30 degrees caudally)
Patient prone head lowered	{	8 Lateral in face down position
		9 Special lateral for posterior part of third ventricle aqueduct and fourth ventricle



ventricles. It is claimed that this can be avoided by injecting the first 5 c.c. (or even 10 c.c.) of air quietly before any fluid has escaped from the lumbar puncture needle.

In cases of traumatic epilepsy it is necessary to study the subarachnoid space as well as the ventricular system in order to search for local dilata-tions or obliterations of the space over an area of cortical scarring. For this purpose larger amounts of air are needed than in cases where tumour being suspected it is only desired to study the ventricles. Amounts of air up to 150 c.c. may be required. Air in this amount almost invariably gives rise to considerable headache, vomiting and sweating. After the injection discomfort continues with diminishing intensity for two to three days, being aggravated by movement of the head.

The indications for encephalography and the danger of using it in cases of raised intracranial pressure have already been mentioned.

Air is absorbed quickly from the subarachnoid space and when there is no obstruction it disappears from the ventricles completely within three to four days.

**Cisternal Encephalography**—In some clinics air is injected by cisternal puncture instead of by lumbar puncture since in this way the air is more likely to go directly into the ventricles. This is a much less painful procedure than lumbar encephalography. It has been clearly shown that the pain of encephalography arises from air in the subarachnoid space and not from air in the ventricles. But though it has become a common procedure for spinal lipiodol tests, cisternal puncture carries a certain small risk of puncture of an artery in the cistern, a danger from which lumbar puncture is free.

### CONTRAST MEDIA

Oxygen has been used in preference to air in the hope that it might be less irritating and more quickly absorbed than air. According to *Davidoff* and *Dyle* there is no striking difference either in the immediate or delayed reaction.

**Positive Contrast Media** including lipiodol, thorotrast and abrodil have all been used especially in displaying the third and fourth ventricles and the aqueduct of Sylvius. Very good pictures have been obtained in this way but even when operation is performed immediately it is difficult, if not impossible, to remove the opaque material and all the substances so far employed have been shown to damage the walls of the cerebrospinal pathways with a tendency to the production of delayed hydrocephalus worse than that for which the patient sought treatment. With improved technique it is becoming increasingly easy to study the third and fourth ventricles with air, a substance which though it gives rise to reactions immediately after injection does not produce any permanent damage of the ventricular ependyma.

## PATHOLOGICAL RADIOLOGICAL APPEARANCES

## I TUMOURS AND OTHER EXPANDING LESIONS

Tumours and other expanding or obstructive lesions almost invariably distort the ventricular system, and their position can usually be inferred from radiological study of the ventricles after an injection. A tumour of one cerebral hemisphere usually displaces the ventricular system to the opposite side and tends to obliterate part or whole of the adjacent lateral ventricle. Tumours of the third ventricle, midbrain and pineal region cause symmetrical dilatation of the lateral ventricles and partial or complete obliteration of the shadow of the third ventricle. Tumours of the posterior fossa produce appearances similar to those of tumours of the third ventricle except that the third ventricle also is dilated and its shadow is not encroached upon, in addition, part of the shadow of the fourth ventricle is also sometimes seen perhaps distorted in shape and position. These changes will be examined in detail in the following pages.

It is necessary at this stage to point out that on rare occasions intracranial tumours give normal ventriculograms. This is seen in the early stages of infiltrating gliomas of the cerebral hemispheres (*Pennybacker and Meadows*) it is not therefore always safe to assume in cases of focal symptoms without rise of intracranial pressure that because the ventricles appear to be in their normal position and of normal shape and size, there is no tumour. Normal ventriculograms may also be found in cases of glomatosis and sarcomatosis of the meninges.

The changes that occur in the ventricular system from the presence of expanding lesions are usually not difficult to interpret if the ventricles are well filled with air and if projections are used that demonstrate the whole of the ventricular system. Nevertheless a systematic description is necessary for an understanding of the various pathological processes that contribute to the abnormal appearances in any one case. In expanding lesions the ventricles may be dilated, obliterated or narrowed and displaced. In practically every case there is a combination of these processes. A tumour affects different parts of the ventricular system in different ways. Locally it tends to encroach upon the ventricle producing narrowing or obliteration and to displace it from its normal position while in the distant parts of the ventricular system there is often dilatation.

The emphasis changes from case to case thus in a case of cerebellar tumour dilatation of the proximal part of the ventricular system is the striking feature of the ventriculograms and the local obliteration and displacement of the fourth ventricle is inconspicuous. In a tumour of one frontal lobe on the other hand, the displacement of the ventricular system to the opposite side will be the most striking feature of the ventriculogram but there may be in addition localised encroachment by the tumour on the shadow of the anterior



FIG 87—Antero posterior ventriculogram showing uniform dilatation of the left lateral ventricle without displacement indicating generalised shrinkage of the left cerebral hemisphere. The rounding of the angles of the dilated ventricle is evident when compared with the unaffected side. Child, aged 7. Attacks of motor epilepsy affecting the right side since infancy. Case No 650.

tion of normal angular contours. In practice the radiologist is not often asked to say whether ventricles are normal or slightly dilated, more often when the ventricular system is on the large size of normal it is for the clinician to say whether the appearances are an early sign of tumour or whether they are due to generalised shrinkage of brain tissue from an old or recent degenerative or atrophic lesion.

horn, diffuse narrowing from oedema of the remainder of the corresponding lateral ventricle, and dilatation of the opposite lateral ventricle.

(1) Dilatation—This is usually due to obstruction of the outflow of cerebro-spinal fluid. The earliest radiological sign of dilatation of the ventricles is a rounding of the margins of shadows that are normally angular. For example, in the lateral ventricle in the antero-posterior view the upper outer angle of the shadow becomes rounded (Fig 87). The distinction between ventricles that are normal and ventricles that are slightly dilated rests largely upon this altera-



FIG 88—Lateral ventriculogram showing severe dilatation of all parts of the lateral ventricles except the descending horns. Same case as Fig 87.

In obstructive lesions of the third and fourth ventricles dilatation of the lateral ventricle is usually most pronounced in the anterior horn and body the extent to which the posterior and descending horns dilate varies greatly from case to case some times there is surprisingly little dilatation of the descending horns when other parts of the lateral ventricle are grossly dilated (Fig 88) In obstructions of long standing the dilatation may reach a considerable degree (Fig 89) The greatest dilatation is seen in cases of infantile hydrocephalus when the lateral ventricles may be so large that the brain substance is less than 1 cm in width (Fig 90)



FIG 89 Antero posterior ventriculogram showing symmetrical dilatation of the lateral ventricles Colloid cyst of the third ventricle Case No 2793



FIG 90—Gross dilatation of the ventricular system A case of infantile hydrocephalus Female aged 9 weeks Case No 203

Generalised dilatation of all the ventricles indicates a lesion at the foramen of Magendie or hydrocephalus from arachnoidal adhesions or from obstruction of the venous sinuses In such cases the dilatation is greatest in the lateral ventricles and least in the fourth ventricle In cases of symmetrical dilatation of the lateral and third ventricles the important radiographic sign is demonstration of the aqueduct of Sylvius if that can be seen filled with air throughout the whole of its course the lesion is localised in the posterior fossa failure to demonstrate the aqueduct throughout the whole of its course suggests that the lesion may be in the midbrain or pineal region in which case the operative

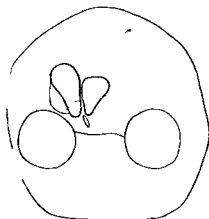


FIG 91—Anteroposterior ventriculogram from a case of meningioma of the left lesser wing of the sphenoid. Both lateral ventricles are dilated, the right more than the left Case No 38a

approach is quite different from that employed for tumours of the posterior fossa. But if the ventricular system has not been well filled with air interpretation should be cautious and the final decision as to the site of operation should rest mainly on the clinical signs. Symmetrical dilatation of the lateral ventricles with partial or complete loss of the shadow of the third ventricle indicates a tumour of the third ventricle or adjacent parts. Asymmetrical dilatation of the lateral ventricles of moderate degree is found with basal tumours notably meningiomas of the olfactory groove and sphenoidal ridge (Fig 91) with tumours of the basal ganglia with multiple metastatic tumours and also on occasions with any supratentorial tumour.

Dilatation of one lateral ventricle is most commonly due to a tumour of the opposite cerebral hemisphere.<sup>1</sup> In such a case the dilated ventricle is dis-

<sup>1</sup> Dilatation of the contralateral ventricle in tumour of one cerebral hemisphere is obviously due to obstruction of the outflow of its cerebrospinal fluid, but it is not always easy to determine the site and cause of the obstruction. The obstruction may be at the foramen of Monro from obliteration of the third ventricle by pressure or from invasion of it by tumour; in these cases the third ventricle is not seen in ventriculograms. In other cases especially in deep seated temporal tumours the obstruction may be at the aqueduct of Sylvius from oedema or neoplastic invasion of the midbrain. In tumour of the cerebral hemisphere obstruction can also be produced at the foramen of Magendie on rare occasions by gross herniation of the cerebellar tonsils through the foramen magnum and it must not be forgotten that in malignant disease with metastases a tumour



FIG 92—Anteroposterior ventriculogram from a case of right temporal abscess showing dilatation and displacement to the left of the left lateral and third ventricles. No air has reached the right lateral ventricle Case No RI 3065

placed towards the healthy side (Fig. 92). Dilatation of one lateral ventricle only is also seen in cases of third-ventricle tumour with blockage of both foramina of Monro, when air has been injected on one side only and the septum lucidum is intact. The ventricle in such a case is not displaced away from the middle line; the septum lucidum bulges towards the opposite side in a manner that is quite characteristic (Fig. 93).

Localised dilatation of one part of the lateral ventricle is seen when the more proximal part of the ventricle is obliterated by tumour (Fig. 94). A part of the lateral ventricle may also become dilated, from tumour growing in or expanding into it (Fig. 95), but not totally occluding it, in such cases the shadow of the dilated part is usually fainter than that of the rest of the ventricle and often irregular in outline. It should be borne in mind that generalised and localised ventricular dilatation occurs also with cerebral atrophy and scarring, as will presently be described.

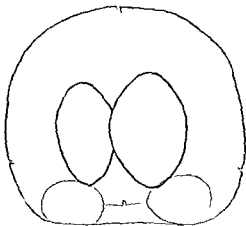


FIG. 93.—Bilateral hydrocephalus in a case of epidermoid cyst of the third ventricle. The septum lucidum is strongly convex to the right, indicating that there is no free communication between the two lateral ventricles, and that the tension of air is greater in the left lateral ventricle than in the right. Case No. 762.

#### (2) Narrowing or Obliteration.—

These changes may affect the ventricular system as a whole, one lateral ventricle only, or a part only of one lateral ventricle.

Generalised obliteration of the ventricular system has never been seen by us, but failure to get air into small ventricles is not uncommon. The ventricles may be so small at times that only a few drops of fluid may be obtained when each lateral ventricle is tapped. In such cases it may be difficult to get adequate filling of the ventricles with air, but if the operator is patient and replaces each drop of fluid with an approximately similar amount of air surprisingly good pictures of the ventricular system can often be obtained.

Generalised narrowing of the ventricular system occurs in some acute

of one cerebral hemisphere may be associated with a tumour in the cerebellum, which causes obstruction and consequent dilatation of the contralateral ventricle. In yet other cases the obstruction occurs in the subarachnoid space at the tentorial opening, from displacement and oedema, or neoplastic invasion of the hippocampal gyrus of one or both sides. Blockage of the subarachnoid channels of one Sylvian fissure is also sufficient to raise intracranial pressure in the proximal cerebro-spinal pathways, and is the probable explanation of dilatation of the opposite lateral ventricle in some meningiomas of the lesser wing of the sphenoid. In all cases of obstruction distal to the foramen of Monro in hemisphere tumours dilatation of the corresponding lateral ventricle would occur, but for the encroachment on it by the adjacent tumour.



FIG 94—Dilatation of posterior and descending horns of left ventricle as a sequel to obliteration of the body of the ventricle by a calcified tumour Case No 3112

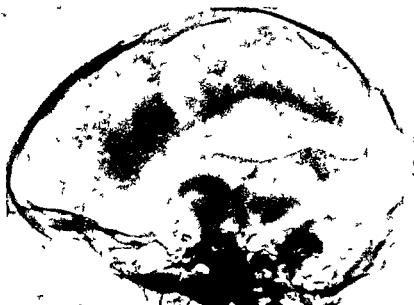


FIG 95—Dilatation of body and anterior part of posterior horn of left lateral ventricle due to a left temporo occipital glioblastoma multiforme The tumour extended from the cortex to the lateral wall of the lateral portion of the ventricle Case No 3300

demyelinating conditions, such as Schilder's encephalitis, and rarely in intracranial tumours. Most tumours of the cerebral hemisphere produce dilatation of the contralateral ventricle, but we have encountered a few cases of frontal meningioma in which the ventricles of both sides were extremely small (Fig 96)

*Obliteration of the shadow of one lateral ventricle* usually indicates that there is a tumour in the corresponding hemisphere which has led not only to great narrowing of the lateral ventricle, but also to partial obstruction of its foramen of Monro. At ventriculography both ventricles are tapped, one is slightly dilated, and the other contains only a drop or two of fluid, or is not found at all. Air is then injected into the patent ventricle, and the ventriculograms show that no air has crossed to the other side, in spite of appropriate manipulations. In such cases the displacement of the dilated lateral ventricle away from the middle line indicates the presence of an expanding lesion on the opposite side, and confirmatory evidence is often seen in displacement of the shadow of the third ventricle (Fig 92). In encephalography, however, the shadow of one lateral ventricle is sometimes absent, even though the ventricles are normal, but in such a case the shadow of the other lateral ventricle is in its normal place and the septum lucidum is in the middle line.

Narrowing of one lateral ventricle is produced by an expanding lesion of the corresponding cerebral hemisphere, such as tumour or abscess. The ventricle is obliterated partly by the expanding lesion, and partly by oedema of the white matter, which may extend throughout the whole cerebral hemisphere (Fig 97). Narrowing of the ventricular shadow that results from oedema does not interfere with the proportions of the ventricle, whereas that which is due to the encroachment of an adjacent tumour is disproportionate (Fig 99a). The more rapid the development of the lesion the more severe, as a rule, is the narrowing of the corresponding ventricle. In meningiomas, which are tumours of slow growth, the ipsilateral ventricle is usually less narrowed and more easily filled with air than in rapidly growing gliomas and in abscesses.

*Localised narrowing or obliteration of one part of a ventricle* occurs with considerable frequency (Fig 98), particularly with tumours that are near or within the ventricle. The curved margins of the narrowed or obliterated zone usually give a fairly accurate indication of the size of the tumour. The



FIG 96—Small lateral ventricles in the presence of a frontal meningioma. Case No 1358



greater the deformity of the body of the ventricle the more likely is the tumour to be deep seated than superficial

With narrowing there occur other alterations of contour In tumours of the cerebral hemisphere the upper lateral angle of the body of the lateral

ventricle on the side of the lesion as seen in antero posterior projection is almost invariably altered as *Lysholm* has pointed out in tumours high up on the convexity this angle is often rounded while in more basally situated tumours it is usually very acute and associated with severe indentation of the lateral wall of the ventricle (Fig 99)

(3) Displacement—Expanding lesions of one cerebral hemisphere tend to displace the whole supratentorial part of the ventricular system to the opposite side The lateral displacement always affects the anterior part of the lateral ventricles especially the bodies more than the posterior parts This is doubtless due to the fact that the posterior part of the cerebral hemisphere is protected from lateral displacement to some extent by the strength and depth of the falx cerebri and by the union of the falx with the tentorium cerebelli The anterior half of the falx cerebri on the other hand is too shallow to prevent lateral displacement of the medial parts of

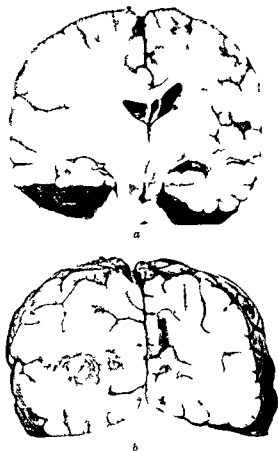


FIG 97 (a and b) Edema of left frontal lobe (a) with narrowing of the left lateral ventricle in a case of small abscess of the left occipital lobe (b) Note also in (a) cavum septum pellucidum Case No 338

one cerebral hemisphere into the dural compartment of the other the unattached edge of the falx also can be displaced laterally

Lateral displacement of the ventricular system is best studied in antero posterior ventriculograms which show clearly the bodies of the lateral ventricles the intervening septum lucidum and the third ventricle Care should be taken to guard against rotation of the head for in pictures that are not in the true sagittal plane the third ventricle may sometimes appear to be displaced

laterally in relation to the anterior horns of the lateral ventricle, which lie nearer the X ray tube

The lowest part of the third ventricle is relatively fixed—it is part of the base



FIG. 98.—Disproportionate narrowing of anterior horn and body of left lateral ventricle due to intra- and para-ventricular left frontal oligodendroglioma. Case No. 7281

of the brain which is anchored by its attachment through the various cranial nerves to the base of the skull, and the displaced ventricular system thus pivots on this point. The type of lateral displacement will vary in some respects with

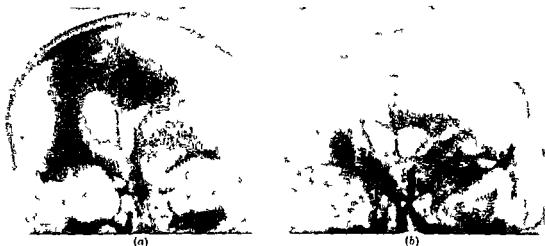


FIG. 99.—Variations in shape of the upper lateral angle of the body of a compressed lateral ventricle. (a) Left temporal ependymoma—the upper angle of the left ventricle is acute and the lateral wall is indented. (b) Left frontal parasagittal subarachnoid cyst—the upper lateral angle is rounded. Cases No. 531 and 971

the position of the tumour in the cerebral hemisphere. The septum lucidum is displaced from the middle line to the greatest extent in parasagittal tumours while the maximum displacement of the third ventricle is seen in tumours of

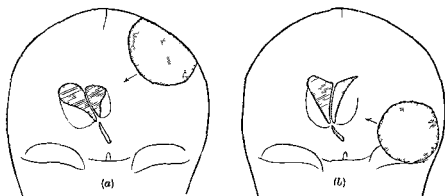


FIG 100 (a and b) —Diagram to show variations of the lateral displacement of the ventricular system with tumours in different parts of the hemisphere

the temporal lobe. Thus if the tumour is in the superior part of the cerebral hemisphere (frontal parietal and occipital tumours) the shadows of the displaced septum lucidum and third ventricle will tend to form a straight line (Fig 100a) but if the tumour is in the inferior part of the hemisphere (temporal and other para Sylvian tumours) the shadows of the septum lucidum and third ventricle form an obtuse angle with one another or even a curve (Fig 100b).

Tumours lying in the superior part of a cerebral hemisphere tend to depress the body of the corresponding ventricle as well as to displace it laterally. Interpretation on this point is complicated however by the fact that tumour of any part of the cerebral hemisphere can cause displacement of the corresponding callosal gyrus beneath the free edge of the falx cerebri (Figs 101 and 70), the displacement may be so pronounced that the body of the lateral ventricle on the side of the lesion is also involved with the production of a ventricular



FIG 101 —Postero anterior ventriculogram from a case of right temporal glioblastoma multiforme. The body of the narrow right lateral ventricle is displaced across the middle line and is depressed by the overlying right callosal gyrus. Case No 2638

deformity that is directly related, not to the position of the tumour, but to the free edge of the *falx cerebri* and the herniated callosal gyrus (Fig 102) Failure to appreciate this mechanism may lead to the conclusion that the body of the ventricle is flattened by a tumour lying above it when the tumour is actually in a remote part of the hemisphere

So far we have considered only lateral displacement of the body of the lateral ventricle and of the third ventricle these being the most important displacements from the ventriculographic point of view But lateral displacement, and displacement in other planes, can occur in any part of the ventricular system The greatest range of displacement is seen in the posterior and descending horns of the lateral ventricles These localised ventricular displacements will be considered in relation to tumours of different regions

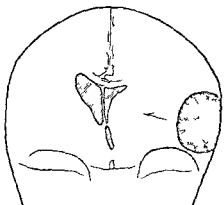


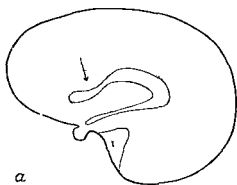
FIG 102—Diagram to show displacement of the left callosal gyrus beneath the free edge of the falx in a case of left temporal tumour

## TUMOURS IN DIFFERENT REGIONS

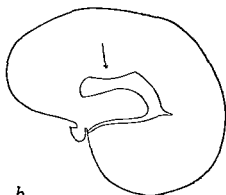
**Frontal Tumours**—The frontal lobe extends from the supraorbital ridge to a line approximately 2 cm behind the groove for the anterior branch of the middle meningeal artery, and thus does not correspond accurately either with the frontal bone or with the anterior horn of the lateral ventricle The most important projections for the study of ventriculograms of frontal tumours are the usual antero posterior projections, a lateral projection with the face upwards, and the standard lateral projection

Gliomas of almost all varieties are common in the frontal lobe, and a not inconsiderable number of them extend into the genu of the corpus callosum as can sometimes be recognised in ventriculograms Meningiomas are common, especially parasagittally at the level of the coronal suture, and also on the base, attached to the lesser wing of the sphenoid or to the cribriform plate and olfactory groove Both frontal lobes are involved in meningiomas of the olfactory groove, as a rule, and in certain glomatous tumours

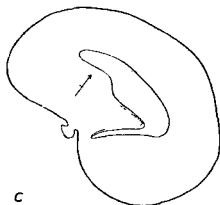
The ventriculographic appearances vary with the position of the tumour, but in all except the bifrontal tumours there is general displacement of the ventricular system from the side of the tumour, and in all the anterior horn of the corresponding lateral ventricle is flattened or obliterated In posterior frontal tumours the body of the ventricle is similarly affected Line drawings of ventriculograms of frontal tumours of differing situations (Fig 103 a-f) show that, in addition to lateral displacement, the anterior horn may be displaced upwards or downwards, it may be indented from above, below, or from the front, or even obliterated As a rule, interpretation is not difficult



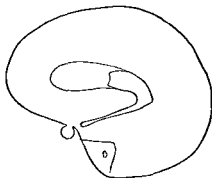
a



b



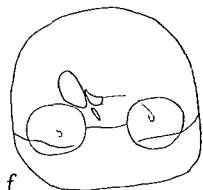
c



d



e



f

FIG 103 —Tracings of ventriculograms from cases of frontal tumour (a and b) Superior frontal tumours (c) Inferior frontal tumour (d and f) Huge frontal tumour with complete obliteration of anterior horn (e) Bifrontal tumour—a meningioma of the anterior end of the falx cerebri

if the ventricles are fairly well filled with air and the appropriate projections are made. Normally in the antero posterior projection without tilt the triangular shadow of the lateral ventricle is seen to be divided horizontally into an upper dark zone representing the whole length of the ventricle seen end-on and a lower less dark area caused by the shadow of the floor of the anterior part of the body of the ventricle seen through the anterior horn (Fig 77). If the body of the ventricle is obliterated the triangular shadow loses its upper translucent zone and becomes homogeneous.

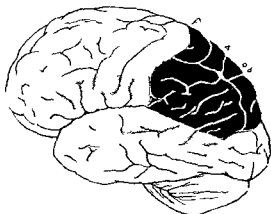


FIG 104—Diagram of parietal lobe

In bifrontal tumours interpretation may be difficult because of the absence of lateral displacement but flattening and other disturbances of the normal contours of the anterior horns can usually be seen especially in lateral projections of the anterior part of the ventricular system (Fig 103f). It is important to remember that as a result of oedema the anterior horns and bodies of the lateral ventricles may be laterally displaced by tumours at distance but in such cases the proportions of the frontal horn are not disturbed.

**Parietal Tumours**—The reader will recall that the greater part of the parietal lobe lies above the level of the lateral ventricle (Fig 104). The



FIG 105



FIG 106

FIGS 105 and 106—Anterior posterior and lateral ventriculograms of a glioblastoma multiforme of the left parietal lobe. Case No 3274

parietal lobe extends from a line approximately 2 cm behind the anterior meningeal groove to a point just anterior to the parieto occipital suture. The most important projections for the study of parietal tumours are the posterolateral, the antero-posterior with 30 degrees tilt cranially and the latero-anterior.

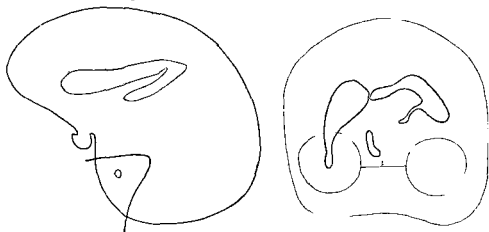


FIG 107 Lateral and postero-anterior ventriculograms from a case of left temporo-occipital astrocytoma Case No 788

The common tumours of the parietal lobe are gliomas and meningiomas the latter usually being parasagittal in situation and often very large. Parietal tumours frequently affect adjacent parts of the brain and are then parieto-occipital, parieto-temporal or parieto-frontal rather than purely parietal in situation.

The whole of the ventricular system is displaced to the opposite side and the ventricle on the side of the lesion is flattened and displaced downwards (Figs 105 and 106). Its body may be forced along with the callosal gyrus under the free edge of the falx producing an appearance in the postero-anterior projection as seen in Fig 102. This occurs more commonly with laterally placed parietal tumours than with parasagittal parietal tumours and as has been described above it also occurs with tumours at a distance from the parietal lobe in parietal tumours. The upper angle of the body of the lateral ventricle is usually rounded. In tumours of the medial surface of the parietal lobe the posterior part

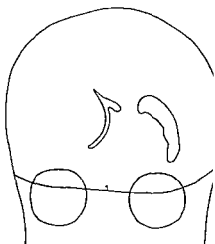


FIG 108 Postero-anterior ventriculogram from a case of occipito-temporal meningioma attached to the dura in the region of the lateral sinus Case No 16

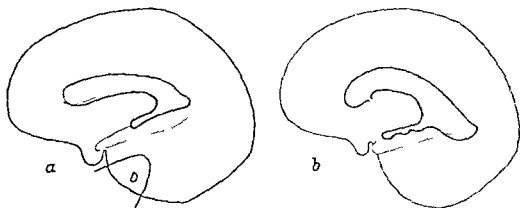


FIG. 109.—Tracings illustrating alterations in the descending horn produced by tumour of the temporal lobe. Cases No. L. H. 40015 1937 and 30700 1937.

of the body and the posterior horn of the lateral ventricle may be displaced laterally from the middle line. Paraventricular parietal tumours occasionally produce dilatation of the posterior part of the body, but the shadow is fainter than normal (Fig. 95). The temporal horn is usually in its normal position.

**Occipital Tumours**—The occipital lobe extends from the posterior end of the parietal lobe to the region of the external occipital protuberance. The most important projections are the postero-anterior, the occipito-frontal, the lateral stereoscopic and the lateral in the face-down position. The asymmetry of the posterior horns of the lateral ventricles that exists in some normal subjects does not present difficulties of interpretation if it is borne in mind that lateral displacement of the ventricular system

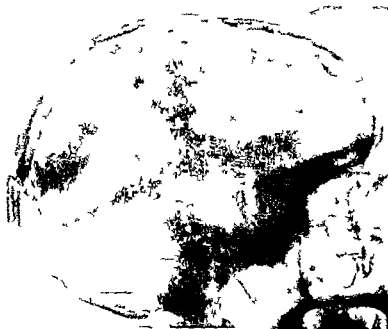


FIG. 110.—Upward displacement and shortening of the descending horn of the lateral ventricle in a case of meningioma of the lesser wing of the sphenoid. The tumour extended into the sella turcica producing overgrowth of the dorsum sellae and other parts.

A youth aged 21 who also suffered from von Recklinghausen's neurofibromatosis. Case No. 564.



always occurs with occipital tumours, but is never found in normal subjects. Tumours limited to the occipital lobe are rare, and the most common are gliomas of all types, and meningiomas, tumours extending into the occipital lobe from the temporal or parietal lobes are much more common, and occasionally meningiomas arise from the tentorium which indent both the occipital lobe and the cerebellum and produce ventriculographic appearances that combine the displacements of occipital tumours with the changes of hydrocephalus.

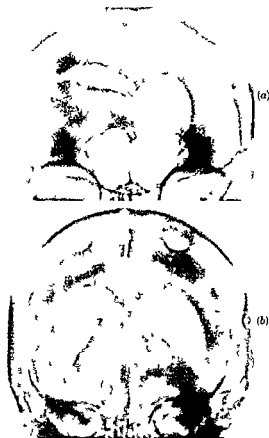


FIG 111—Tumours of the basal ganglia  
(a) Gross filling defect of the floor of the right lateral ventricle. Case No. R11005  
(b) Tumour of left thalamus. There is slight filling defect of the floor of the body of the left lateral ventricle. The third ventricle as seen in this postero-anterior projection is displaced towards the right and both lateral ventricles are dilated. Case No. 3228

greatest value in ventriculograms of temporal tumours are the antero-posterior, the postero-anterior, the lateral stereoscopic, and the lateral projection in the face up position. Tumours in the temporal lobe are very common and are for the most part gliomas, by far the greatest number being glioblastomas multiforme. Meningiomas of the middle fossa, cavernous

In occipital tumours there may be obliteration of the posterior horn as seen in the lateral view in the face down position or there may be narrowing and very great displacement of the posterior horn in any direction. With tumours that are parietal as well as occipital the postero-anterior views may show downward displacement of the trigone and of the posterior part of the descending horn. When the tumour is situated in the lower part of the occipital lobe as in occipital extension of a temporal tumour, the posterior horn is displaced upwards together with the posterior part of the body and the posterior part of the descending horn (Figs 107 and 108).

**Temporal Tumours**—The temporal lobe lies below a line extending from the external angular process of the frontal bone to the parietal eminence. The projections of

sinus, posterior edge of the lesser wing of the sphenoid and of the para Sylvian region also occur these often give characteristic appearances in ordinary radiograms

Temporal tumours produce great lateral displacement of the ventricular system especially of the third ventricle which usually forms with the septum lucidum an angle or curve convex towards the opposite side (Fig 100) The descending horn is obliterated or is thinned (Fig 109) and displaced most usually in a medial and upward direction (Fig 108) and sometimes to a remarkable degree (Fig 110) In posterior temporal tumours the anterior part of the temporal horn may be filled while the posterior part only is narrowed

When the tumour is large there is often compression and narrowing of the body of the corresponding lateral ventricle its upper outer angle becomes pointed and its lateral aspect is concave in contradistinction to the appearance seen with parietal tumours when the upper outer angle of the ventricular body is rounded and its lateral margin straight or even convex outwards (Fig 99) The lateral displacement may be so great that the callosal gyrus is displaced beneath the falx with the production of further



FIG. 11. —(a) Astrocytoma of the postero-inferior part of the left frontal lobe projecting into the septum lucidum and into the lateral ventricle. Case No 983



FIG. 11. (b) Calcified oligodendroglioma of optic thalamus projecting up into the body of the lateral ventricle. It is seen that the actual size of the tumour cannot be estimated from the size of the area of calcification. Case No 2509

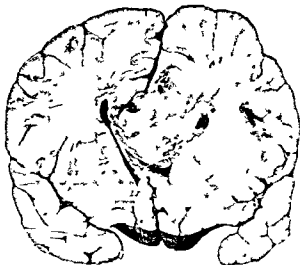


FIG 113—Glioblastoma multiforme of the corpus callosum Case No 1874

associated with displacement of the third ventricle to the opposite side. This deformity is best seen in antero posterior, postero anterior and lateral stereoscopic projections (Fig 111). When the defect in the floor of the lateral ventricle forms the large arc of a small circle (Fig 112*b*) it may be difficult to tell by ventriculography whether the tumour is primarily intra ventricular and operable, or whether it arises from the basal ganglia extending secondarily into the ventricle and is thus inoperable.

**Tumours of the Corpus Callosum and Septum Lucidum**—Malignant gliomas arise in the corpus callosum or spread into it from the white matter of one or other hemisphere. The corpus callosum increases in depth and is displaced downwards on to the bodies of the lateral ventricles tending to push them apart (Fig 113). In

ventricular deformity (Figs 100 and 101).

**Tumours of the Basal Ganglia and Caudate Nucleus**—These deeply placed tumours, almost invariably gliomas of rapid or slow growth sometimes produce appearances of bilateral hydrocephalus with no filling of the third ventricle, that are difficult to distinguish from the appearances of tumours within the third ventricle. More often however, there is a characteristic deformity of the floor of the body of the corresponding lateral ventricle,



FIG 114—Antero posterior projection of the same case as Fig 113

the antero posterior ventriculogram the lateral ventricles appear to be separated from one another and their upper margins are pointed (Fig 114) but no actual separation occurs unless the tumour spreads into the septum lucidum. The lateral ventricles are usually not dilated if the tumour occupies the centrum semiovale of one side as well as the corpus callosum they will be displaced towards the opposite side.

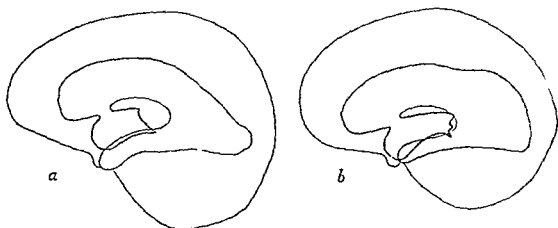


FIG. 115.—Tracings of ventriculograms. (a) pineal tumour. (b) stricture of aqueduct of Sylvius. Cases No. 364 and 1359.

Gliomas limited to the septum lucidum have been encountered some of them benign astrocytomas. They give rise to bilateral symmetrical hydrocephalus and obstruction of the foramina of Monro with failure of the third ventricle to fill. The shadow of the tumour is visible otherwise the appearances are identical with those of tumour of the third ventricle.

**Tumours of the Third Ventricle and Midbrain.**—The position of the third ventricle and the special projections necessary to display it in ventriculograms have already been described. The importance of completely filling the lateral ventricles with air in order to secure good pictures of the third ventricle cannot be over-stressed. Of the tumours that occupy the third ventricle region some arise within the ventricle—colloid cysts, epidermoid cysts arising at a high level and cholesteatomas—and are benign operable tumours. Others arise in its wall or in the optic chiasm and are inoperable gliomas and others extend from adjacent structures. From the pituitary gland and its stalk epidermoid cysts or pituitary adenomas may expand into the region of the anterior part of the third ventricle and large suprasellar meningiomas produce similar deformity. Tumours of the pineal body extend into the posterior part of the third ventricle or even fill the whole of its cavity. Tumours of the basal ganglia also obliterate the cavity of the third ventricle.

In tumours of the anterior part of the third ventricle there is obstruction of the foramina of Monro and bilateral hydrocephalus of the lateral ventricles.

In the majority no air is seen in the third ventricle though repeated strong hyperextension of the neck and gentle shaking of the head from side to side will sometimes coax air into the anterior part of the third ventricle when the first pictures have shown no air in that region. The ventriculograms of colloid cysts of the third ventricle are often characteristic (Fig 144) because of the rounded outline of the shadow and its position immediately below the foramina of Monro but those of the other tumours of the anterior part of the third ventricle are not in any way distinctive except that lateral displacement of a partially filled third ventricle indicates that the tumour arises in the basal



FIG. 116.—Forward displacement of the aqueduct of Sylvius in a case of cerebellar medulloblastoma. Case No. 968.

ganglia. In most cases evidence of the pathological nature of the tumour is not provided by the ventriculogram and can be derived only from clinical examination or from operation.

Tumours of the posterior part of the third ventricle are mostly tumours of the pineal body or midbrain. It is important that every effort should be made to distinguish them from tumours of the anterior part of the third ventricle since the operative approach is quite different. The distinction is not difficult except in tumours that fill the whole third ventricle when it may be impossible to decide by ventriculography whether the tumour has arisen in the region of the hypophyseal stalk or in the pineal region. Tumours of the

pineal body and midbrain, and strictures of the aqueduct of Sylvius, often give the same clinical syndrome (signs of raised intracranial pressure, pupils that are fixed to light, and loss of upward movement of the eyeballs), and some cerebellar tumours can imitate pineal tumours closely in their clinical signs. This group of cases tests the technique of ventriculographic examination more than any other in them it is of the utmost importance to fill and display the posterior part of the third ventricle and the aqueduct of Sylvius. The lateral ventricles are symmetrically dilated and the anterior part of the third ventricle as a rule filled. In pineal tumours the shadow of the posterior part of the



FIG 117.—Lateral displacement of the aqueduct of Sylvius and fourth ventricle in a case of haemangioblastoma of the left cerebellar tonsil. Case No. L.H. 30459/1938<sup>1</sup>

third ventricle is almost invariably encroached upon, and the aqueduct of Sylvius, if it shows at all, is narrowed and displaced downwards (Fig 115a). In tumours of the midbrain, according to *Lysholm*, the narrowing and displacement of the aqueduct, in the absence of any encroachment on the shadow of the posterior part of the third ventricle, provide a pathognomonic picture. In *strictures of the aqueduct of Sylvius* the third ventricle is grossly dilated and the shadow of the aqueduct is curtailed (Fig 115b), or invisible. The visible part of the aqueduct may be trumpet shaped, it is never displaced forwards or laterally, as in cerebellar tumour. If the whole of the aqueduct is seen the

<sup>1</sup> This case and the cases of Figs 153, 171, and 174 were under the care of Mr D. W. C. Northfield to whom we are indebted for generously placing his material at our disposal.



FIG 118—Hydrocephalus due to blockage of the foramina of Luschka and Magendie, as a result of chronic adhesive meningitis. The fourth ventricle is marked by arrows. In front of that are seen the anterior parts of the descending horns the third ventricle, and the anterior horns each with its own fluid level. Case No R I 3893

assumption should be that the lesion is in the posterior fossa, even though no part of the shadow of the fourth ventricle is visible

**Cerebellar Tumours**—The fourth ventricle is best examined by lateral,

both postero-anterior, and special projections (page 85). The common tumours of the cerebellum are gliomas (especially astrocytoma, medulloblastoma and ependymoma), haemangioblastomas, tuberculomas, metastatic tumours and cholesteatomas. Cerebellar tumours are prevented from expanding upwards by the tentorium, and in consequence do not displace the posterior horns of the lateral ventricles. Ventriculograms show dilatation of both lateral and third ventricles and of the aqueduct of Sylvius. The aqueduct may be displaced



FIG 119—Cystogram of sellar epidermoid (Rathke pouch) cyst extending into the right frontal lobe. There is also calcification within the sella and separation of the coronal suture. Boy aged 6. Case No 188

forwards towards the basilar occiput producing a kink (Fig 116) and is at times bent laterally (Fig 117). The fourth ventricle is obliterated in part or occasionally completely and may be displaced laterally when the tumour occupies the lateral cerebellar lobe rather than the vermis (Fig 117). *Lysholm* states that in all his cases of tumour of the lateral cerebellar lobe in contrast to tumours of the vermis it was possible to demonstrate air in the fourth ventricle.

**Pontine Tumours**—The aqueduct of Sylvius and the fourth ventricle are displaced in a dorsal direction and at times laterally. In some cases the proximal part of the aqueduct is dilated.

### ARACHNOIDITIS

Adhesions and thickening of the arachnoid in the posterior fossa always give rise to great dilatation of the whole ventricular system but especially of the fourth ventricle. Usually the blockage occurs at the foramina of Luschka and Magendie but at times dense adhesions both subdural and subarachnoid are found at the foramen magnum. If the foramina of Luschka and Magendie are obliterated the dilatation ends at this region (Fig 118), but with obstruction at the foramen magnum the air may be seen to pass out into the cisterns especially into the cisterna magna. There is no displacement of the fourth ventricle and aqueduct.

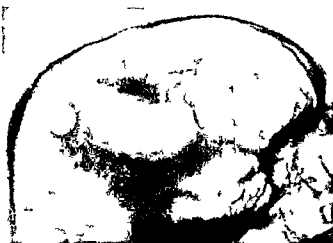


FIG 10—Cystogram of left fronto-parietal cystic tumour. Arrows mark the probable site of a mass of solid tumour in the wall of the cyst. The calcified patch below the cyst is probably also part of the solid tumour. Case No. 3377.

### CYSTOGRAPHY

During ventriculography for unlocalised brain tumours the needle seeking the lateral ventricle sometimes enters a cyst. Such cysts are with scarcely an exception associated with a solid tumour in the form of a mural nodule. While the cyst is being emptied air can be injected or if the cyst is large, air will enter spontaneously through and alongside the needle as more and more fluid is withdrawn since the brain is enclosed in a bony case that will not collapse (Fig 119). Appropriate projections should be employed to show the medial lateral anterior and posterior walls of the cyst outlined by air. The



extent and position of the cyst can thus be accurately shown, and sometimes the position of the mural nodule is disclosed by a flattening of, or projection into one part of the spherical or ovoidal border of the cyst (Figs 120 and 121)



FIG 121—A glioblastoma multiforme of the left optic thalamus and third ventricle (a) Is a cystogram of the more superficial part of the tumour (b) A ventriculogram shows a defect in the shadow of the lateral ventricle corresponding to the cyst and another large defect in the posterior part of the shadow of the third ventricle Case No 2141

Similar technique may be employed to display the precise situation of large abscesses after pus has been withdrawn from them. In abscess thorotrast can be used instead of air with reasonable safety, as suggested by Kahn (Fig 122)

## II SCARRING AND ATROPHIC LESIONS

Destruction or shrinkage of brain tissue from any cause is followed by compensatory dilatation of the cerebrospinal fluid pathways which can be displayed by encephalography or ventriculography. Thus as was first shown by *Dandy Foerster* and *Penfield*, a large number of contracting and atrophic lesions of the brain can be studied by air injection (Fig 87). The shrinkage of the brain may be localised as for example in penetrating head injury or porencephaly (Fig 123) or generalised as in diffuse degenerative lesions of the brain, like *Alzheimer's disease*.

From the practical standpoint the most important of the contracting lesions are those which follow head injury. After a penetrating head injury the dura becomes adherent to the surface of the brain at the site of injury. The damaged brain tissue is absorbed and there is overgrowth

of the fibrous elements of the neuroglia. The dura like any other mesoblastic connective tissue in response to injury forms fibroblast and these grow into the damaged brain tissue with the result that there is progressive deposition of collagen and scarring and contraction of the damaged brain. The scarred brain is adherent to the dura and to the overlying scalp. The scalp thus becomes sunken through the area of bone defect. At the same time the scarred area of brain tissue exerts a pull on the deeper structures and produces a diverticulum of the adjacent lateral ventricle.

The process of scar formation may go on for years as Penfield has shown with increasing traction on the surrounding brain and increasing liability to epilepsy. The diverticulum of the ventricle becomes progressively larger until nothing remains between it and the scalp at the site of the old penetrating injury except a thin layer of dense collagen and neuroglia containing perhaps a few



FIG. 13.—A large porencephalic cyst of the left occipital lobe connected with the lateral ventricle. Male aged 13. Weakness of right hand noted in the first year of life. From age of 7 epileptic attacks beginning in right hand. Right lower homonymous hemianopia. Motor and sensory signs in right arm and leg. Case No. 9466.



FIG. 14.—Cephalogram illustrating a large porencephalic cyst of the left temporal lobe extending to the thalamus. Case No. 9846.

degenerated nerve cells. Wide excision of such a scar with consequent release of traction on the brain is followed in a considerable number of cases by relief from epileptic attacks.

Such cases come under the purview of the radiologist when epileptic attacks have supervened at a variable interval of years after the head injury. Encephalography is the method of choice for air injection because in this type of case it is not dangerous and there is need to study the subarachnoid space as well as the ventricular system. A large amount of air should be injected up

to 150 c.c., or even more. Projections tangential to the diverticulum as well as routine projections should be employed and vary with each case. The radiograms show a wide mouthed diverticulum extending from the lateral ventricle up to the site of the old bone defect (Fig. 124). The rest of the lateral ventricle on the side of the lesion is slightly or moderately dilated, the ventricle on the opposite side is normal or slightly dilated. The septum lucidum lies vertically in the middle line or is slightly displaced towards the side of the



FIG. 124.—Fraction diverticulum of left lateral ventricle in a case of traumatic epilepsy (compound left frontal injury at the age of 6. Epilepsy from the age of 16 onwards. Operation at the age of 29, no fits thereafter up to the time of last observation five years later. Case No. 1339.)

lesion. In addition there may be some collections of air on the surface of the brain around the edge of the bone defect.

Closed head injuries may also produce focal destruction of brain tissue with changes and effects similar to those found after penetrating injuries. There is in these cases as would be expected less invasion of the brain by collagenous fibrous tissue than in penetrating injuries and in consequence the ventricular diverticulum tends to be smaller. On the other hand the superficial lakes of subarachnoid fluid may be larger especially in those cases in which the dura has not become adherent to the surface of the brain.

Focal scarring of the brain occurs after healing of a brain abscess and may often be associated with ventricular diverticulum and epilepsy. After

removal of tumours of the cerebral hemisphere the resulting cavity is usually filled in part by a diverticulum of the ventricle if during removal of the tumour it has been necessary to divide many cerebral veins or arteries the shrinkage of brain tissue may extend beyond the bed of the tumour. All of these changes can be displayed by encephalography.

Shrinkage of the brain with the formation of ventricular diverticulum or subarachnoid cystic collections follows after an interval thrombosis of cerebral veins obstruction of cerebral arteries and the absorption of intra cerebral blood clot. The area of brain damage is usually more extensive than in focal head injury. Cystic cavities in the brain substances sometimes occur in these cases and may be connected with the ventricle by tiny holes (Fig 125).

Diffuse dilatation of a large part or the whole of one lateral ventricle is found in cases of epilepsy and dementia following birth injury and in congenital and infantile cerebral atrophy the causes of which are not yet clearly defined. In the focal and diffuse cerebral atrophies of adults also localised or diffuse dilatation of the ventricular system will be found.



FIG 125—Porencephalic cystic diverticulum of the right lateral ventricle filled by encephalography. The enlarged and tortuous meningeal channels suggest that it may have been secondary to some angiomatous malformation. Case No 349.

## CEREBRAL ANGIOGRAPHY

Cerebral angiography was introduced by *Fgas Moniz* in 1927. The method consists in injecting radio-opaque solutions into the cerebral circulation and examining the vascular tree with X rays. At first *Moniz* called it "cerebral arteriography" but later he found that the veins and venous sinuses could also be studied and so changed the name. Various solutions have been used sodium iodide (25 per cent) abrodil and thorotrast. The least irritating of these is thorotrast and that is now used exclusively. It is not however an ideal medium for it is a permanently radio active substance and is not eliminated from the body, it is stored in the reticulo endothelial system and can in some cases be shown by radiograms in the liver and spleen months after it has been introduced into the circulation. *Russell* and *Northfield* have shown that thorotrast may also be retained in the cerebral vessels around a tumour where it may aggravate the pre existing symptoms. In spite of

these objections to the use of thorotrast cerebral angiography is in some cases of such diagnostic importance that its advantages outweigh the chances of immediate or remote complications. The method has added considerably to our knowledge of cerebral vascular disorders.

### Technique

Thorotrast is introduced into the common carotid artery under local anesthesia usually after exposure of the artery by open dissection though in a few clinics subcutaneous injection has been practised. The fact that the external carotid and its branches also become filled is not a hindrance to interpretation of the pictures. Moniz has shown that the circulation of opaque solutions is much slower in the external carotid and its branches to the meninges and coverings of the cranium than in the internal carotid which supplies only the brain and the retina. He estimates that the circulation of blood from the internal carotid artery through the cerebral capillaries into the large cerebral veins is accomplished in three seconds.

The injection of thorotrast is made as quickly as possible through a 20 gauge serum needle and 10 c.c. of 25 per cent solution of thorotrast are used. An exposure not longer than one tenth second is made when 6-7 c.c. of the solution have been injected and a second film is taken two seconds later. For this technique cassettes must be changed rapidly and if mistiming is to be avoided it is most important that the surgeon and radiologist should rehearse the procedure several times before each injection is made. Most of the information is obtained from the first film. The carotid artery on the side of the suspected lesion is first injected and after the films have been developed it may be desirable to inject the artery of the opposite side. Both projections are lateral the patient lying on his back.

There are variations of this technique. Moniz uses a special rotating cassette and takes six exposures in quick succession. Lohr and others of the German school take antero posterior exposures as well as lateral. Each new projection entails an additional injection of thorotrast.

A method of injecting the basilar artery has been devised by Moniz but is very seldom used. The injection is made into the subclavian artery after dissection and compression of the artery on the distal side. This causes the thorotrast to be forced back against the blood stream into the vertebral artery and so to the base of the brain.

### Normal Appearances

The arteries of the brain are seen on the film taken two seconds after the commencement of the injection of the thorotrast into the common carotid artery. The internal carotid artery reaches the interior of the skull by passing anteriorly through the petrous portion of the temporal bone in the carotid canal. As it leaves the carotid canal it turns upwards and then describes an

S-shaped loop lying between the layers of the dura mater which form the cavernous sinus. This loop is known as the "carotid siphon" and forms part of the lateral wall of the pituitary fossa (Fig 126). The normal shape of the siphon varies greatly, and a complete extra loop may sometimes be seen. The siphon divides into three main groups of vessels: (i) the anterior cerebral, supplying the frontal lobes and dividing into the pericallosal and callosal mar-



FIG 126 — Normal arteriogram showing needle in the common carotid, the external carotid and some of its branches: the internal carotid and its branches: SG the Sylvian middle cerebral group. AC the anterior cerebral artery. AC', pericallosal branch. Case No. 2216.

ginal arteries, which usually show plainly in the arteriogram, (ii) the Sylvian or middle cerebral group, which is the main group and supplies the bulk of the blood to the hemispheres, it gives rise to the posterior temporal and posterior parietal branches, (iii) a small posterior cerebral group, usually formed by the junction of the posterior communicating artery with the posterior cerebral artery. This group fills after some delay, and is poorly outlined in most cases. Occasionally, however, the main posterior cerebral artery arises directly from the internal carotid instead of from the basilar, and then it shows clearly.

The veins of the brain appear on the film taken two seconds after the first and four seconds after the beginning of the injection and the sinuses of the skull are best seen two seconds later (Fig 127). An intermediate phase may be seen between the first and second exposures in which the thorotrast is in the capillaries and the result is that the whole of the brain field appears more opaque than usual. A fraction of a second later the superficial veins are seen to be filled—the superior cerebral veins running to the superior sagittal sinus and the inferior cerebral veins towards the base of the hemispheres.



FIG 127—Normal phlebogram showing superior and inferior longitudinal sinuses, great vein of Galen at a glance. Case No. 16.

Two large veins are often visible—the first the anastomotic vein of Trolard connects the superior sagittal and the cavernous sinuses—the second the posterior anastomotic vein of Labbe unites the middle cerebral vein (superficial Sylvian) to the lateral sinus. Later still the sinuses of the skull fill but it is uncommon to see good filling of the superior longitudinal and lateral sinuses.

#### Indications and Pathological Appearances

Cerebral angiography has been employed for localisation of intracranial tumours and other expanding lesions of the brain. These lesions displace

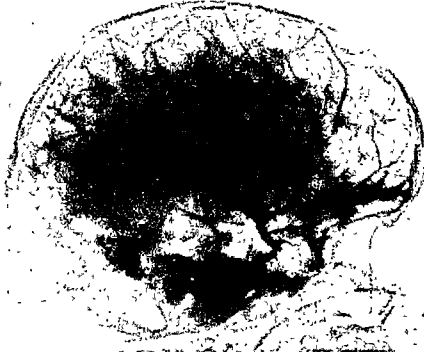


FIG. 128a.—Left arteriogram in a case of right temporal abscess. The vessels run a straighter course and are more widely separated from one another than normal, indicating hydrocephalus of the left lateral ventricle.

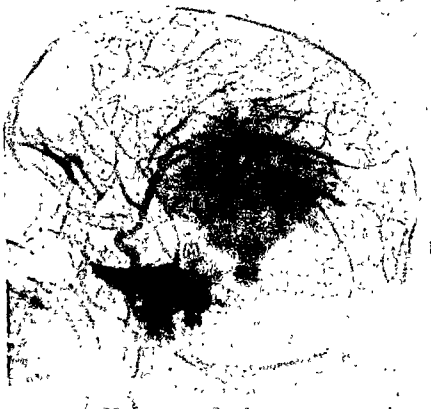


FIG. 128b.—Right arteriogram in a case of right temporal abscess (the same case as Fig. 128a). The main middle cerebral arteries are displaced upwards by the abscess. Case No. 2858.



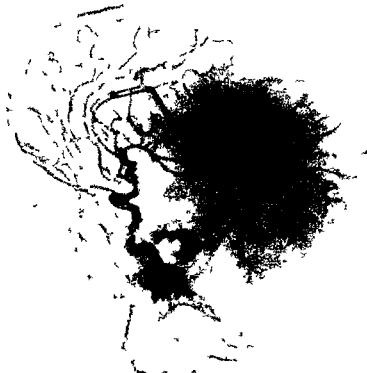


FIG. 128c. Depression of the posterior part of the anterior cerebral artery and its branches by a large parietal and frontal parasagittal meningioma. Case No. 967.



FIG. 129. Small bilobular aneurysm of the right posterior communicating artery. Case No. 2716.

vessels (Fig 128 *a b* and *c*) and retard obstruct or at times cause dilatation of the vessels in the vicinity of the tumour. The circulation within the tumour itself may be visualised and this may provide a radiographic diagnosis of the pathological type of the tumour, as Almeida Lima has shown. The presence of a network of large and irregular venous channels in the area of the tumour suggests a glioblastoma multiforme while a faint diffuse shadow produced by thorotrast within the capillaries of the tumour suggests a meningioma. Absence of any vessels in the region of a tumour, which is recognised by displacement of main arteries as in Fig 128 (*b* and *c*) suggests the presence of a cyst a solid astrocytoma or an abscess.

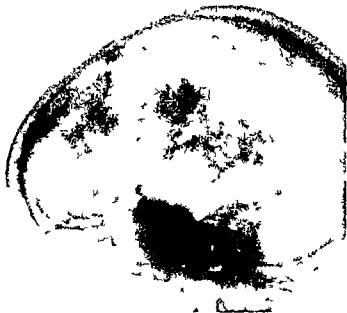


FIG. 130. Large aneurysm of the internal carotid artery. Woman aged 53. Case No. 310.

In view of the possible immediate and remote complications of thorotrast and the efficiency of ventriculography it is questionable whether cerebral angiography should be employed as a routine accessory method of tumour diagnosis until some radio-opaque substance has been found that is both not irritating to the cerebral vessels and not permanently radio active. The clear indications for cerebral angiography are (1) for the localisation of cerebral aneurysms in which surgical treatment is desirable (*Bramwell and Dott*), (2) for the differentiation without operation between aneurysms of the carotid and ophthalmic arteries and pituitary tumours (*Jefferson*), (3) to establish the pathological diagnosis and precise situation of certain angiomatous malformations of the cerebral vessels cases presenting as a rule, under the guise

of intracranial tumours but requiring quite a different method of treatment namely irradiation with or without carotid ligation

**Cerebral Aneurysm** shows usually as a spherical sac attached to one of the main cerebral arteries (Fig 129) After the thorotrast has passed from the cerebral circulation some of it may still remain in the sac of the aneurysm. Large aneurysms are easily recognised (Fig 130) but are occasionally lined with clot so that the size of the thorotrast shadow is not a precise indication of the size of the aneurysmal sac

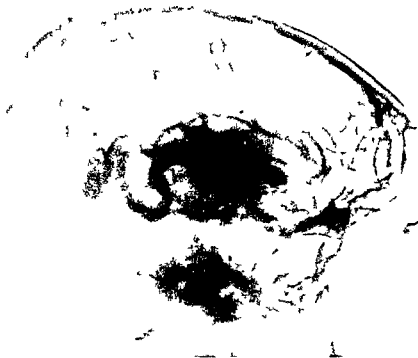


Fig 131 A teroveot angioma of right cerebral hemisphere (Case No 3426)

**Angiomatic Malformations** —The radiographic appearances vary greatly. In arterio venous aneurysm there is usually the shadow of one or more dilated and tortuous vessels leading up to a coiled mass of dilated blood vessels in which there is often some flecks of calcification (Fig 131) Sometimes the area of malformation shows in angiograms only as a diffuse shadow of irregular outline and varying intensity (*Tonnies*) In venous angioma the angiograms show only slight venous enlargements and these are not easy to display

**Frontal Lobe** —Lesions situated anteriorly will press the anterior cerebral and pericallosal arteries backwards and will depress the anterior part of the Sylvian group. The carotid siphon may also appear to be depressed and

forced back towards the petrous bone. The posteriorly placed frontal lesions depress the Sylvian group, causing a concavity in its upper edge, the pericallosal artery is depressed and the carotid siphon displaced downwards, but not posteriorly.

**Parietal Lobe.**—Parietal lesions usually cause the vessels supplying that part of the hemisphere to be crowded towards the base of the skull. If the tumour is situated low in the hemisphere there may be separation of the vessels. The vessels chiefly affected are the posterior branches of the middle cerebral artery, consisting of the ascending parietal, the temporo parietal, and the posterior temporal arteries. If the tumour lies deeply in the brain substance the vessels may be separated from each other and stretched over the tumour.

**Temporal Lobe.**—In lesions of the temporal lobe the commonest finding is an upward displacement and crowding together of the Sylvian group while the carotid siphon tends to lose its normal curves and become straightened. The vessels at the lower edge of the Sylvian group may become concave at their lower margin. If the tumour is situated far posteriorly in the temporal lobe the carotid siphon will be unaffected, but the posterior part of the Sylvian group will be elevated. It is with this group of tumours that arteriography may give assistance in a very difficult case, though it is very unusual to be unable to make a diagnosis from ventriculography.

**Pituitary Region.**—The tumours of the pituitary fossa and neighbouring parts may cause delay in the passage of thorotrast, owing to compression and narrowing of the arterial trunk between the tumour mass and the lesser wing of the sphenoid, at the same time the normal curves of the siphon may be straightened, while in the postero anterior view lateral displacement of the vessel may also be seen.

## BIBLIOGRAPHY

- BALADO, M., "Técnica de la Ventriculografía con Lipiodol," *Arch Argent de Neurol*, 1930, VI, 34, "Anatomía Clínica y Radiológica del Tercer Ventrículo," *La Semana médica*, 1931, XXXVIII (1), 413.
- BALADO, M., and DIMITRI, Y., "Compresiones de Médula y su diagnóstico por el Lipiodol," *Arch Argent de Neurol*, 1930, VI, 1.
- BRAMWELL, E., "Neurology," *Trans Ophthalmol Soc of the United Kingdom*, 1934, LIV, 205.
- CATRNS, H., "Accessory Methods of Diagnosis of Intracranial Tumour," *Trans Med Soc London*, 1935, LVIII, 50.
- CARRILLO, R., "La Radiografía del cuarto ventrículo," *La Semana médica*, 1931, XXXVIII, 11, 719, "Mecanismo de la Evacuación del Lipiodol Intraventricular," *Arch Argent de Neurol*, 1937, XVI, 134.
- DANDY, W. E., "Ventriculography following Injection of Air into Cerebral Ventricles," *Ann Surg*, 1918, LXVIII, 5. "Roentgenography of the Brain after the Injection of

- Air into the Spinal Canal," *Ann Surg*, 1919, LXX, 397; "Localisation or Elimination of Cerebral Tumours by Ventriculography," *Surg Gynec. and Obstet*, 1920, XXX, 329, "Localisation of Brain Tumours by Cerebral Pneumography," *Amer. Journ Roentgenol*, 1923, X, 610, "Pneumocephalus," *Arch Surg*, 1926, XII, 949;
- BENIGN Tumours in the Third Ventricle," 1933, London, Bailliere, Tindall & Cox
- DAVID, M., STUHL, L., ASKENASY, H., and BRUN, M., "Aspects Pneumographiques de l'Aqueduc de Sylvius et du IV<sup>e</sup> Ventricule," *Journ de Rad et d'Elect*, 1937, XXI, 193
- DAVIDOFF, L. M., and DYKE, C. G., "The Normal Encephalogram," 1937, London, Kimpton
- DOTT, N. M., "Intracranial Aneurysms - Cerebral Arteriography," *Ldin Med Journ*, 1933, XL, 219
- DYES, O., "Die Hirnkammerformen bei Hirntumoren," *Fortschr. a d Geb. d Roent genstrahl*, 1937, LII, 11
- DYKE, C., and DAVIDOFF, L. M., "Congenital Absence of the Septum Pellucidum," *Amer Journ Roentgenol*, 1935, XXXIV, 573
- DYKE, C., ELSBERG, C. A., and DAVIDOFF, L. M., "Enlargement of the Defect in the Ar Shadow Normally Produced by the Choroid Plexus," *Amer Journ Roentgenol*, 1935, XXXIII, 736
- FOURSTER, O., "Encephalographie-Erfahrungen," *Ztschr f die gesamte Neurologie u Psychiatrie*, 1924, XCIV, 512
- FOURSTER, O., and PENFIELD, W. G., "Structural Basis of Traumatic Epilepsy," *Brain*, 1930, LIII, 99
- FREEMAN, W., "Effect of Injection of Colloidal Thorium Dioxide on the Ventricles and Subarachnoid Spaces," *Arch Neurol and Psychiat*, 1937, XXXVIII, 340
- FREEMAN, W., SCROENFIELD, H. H., and MOORE, C., "Ventriculography with Colloidal Thorium Dioxide," *Journ Amer Med Assoc*, 1936, CVI, 960
- HELMANN, K., OBRADOR, S., and DOTT, N. M., "Intracranial Aneurysms and Allied Clinical Syndromes - Cerebral Arteriography in their Management," *Lisboa Med*, 1937, XIV, 782
- JOHNSON, V. C., and LIST, C. F., "Ventriculographie Localisation of Intracranial Tumours," (1) Third Ventricle and Thalamus, *Amer Journ Roent*, 1937, XXVIII, 77 (2) The Aqueduct, Pons and Cerebello pontine Angle, *Amer Journ Roent*, 1938, XL, 349
- JUIF, M. H., "Tumours of the Lateral Ventricles and Adjacent Parts," *Proc Roy Soc Med*, 1936, XXI, 1168, "Ventriculography," *Brit Journ Radiol*, 1936 IX, 147
- KAHN, E. A., "Treatment of Encapsulated Abscess of the Brain - Visualization by Colloidal Thorium Dioxide," *Arch Neurol and Psychiat*, 1939, XLI, 158
- LOCKE, C. E., "Studies of Casts of the Cerebral Ventricles," *Arch Neurol and Psychiat*, 1926, XV, 588
- LOHR, W., and JACOBI, W., "Die kombinierte Enzephal Arteriographie," 1933, Leipzig, Thieme
- LIMA, ALFIDA, "Contribuição para o Estudo de Cirulação dos Tumores Intracranianos," 1938, Lisboa, der Silva
- LYSHOLM, E., "Das Ventrikulogramm," I Röntgentechnik, *Acta Radiol*, 1935, Suppl 24 "Das Ventrikulogramm," II Die Seitenventrikel, *Acta Radiol*, 1937, Suppl 25 "Das Ventrikulogramm," III Dritter und Vierter Ventrikel, *Acta Radiol* 1935, Suppl 26, "Neuere Erfahrungen mit der Ventriculographie der 3 u 4 Hirnkammer," *Nervenarzt*, 1937, X, 1, "Radiological Experience in Ventriculography," *Brit Journ Radiol*, 1938, XI, 273
- MERRITT, H. H., and FREMONT SMITH, F., "The Cerebrospinal Fluid," 1937, Philadelphia Saunders Co
- MONIZ, E., "Diagnostic des Tumeurs Cérébrales et epreuve de l'Encéphalographie Artérielle," 1931, Paris, Masson & Cie, "L'Angiographie Cerebrale," 1934, Paris Masson & Cie
- NORTHFIELD, D. W. C., "Cerebral Angiography," *Proc Roy Soc Med*, 1936, XXI, 1172

- NORTHFIELD D W E and RUSSELL, D S The Fate of Thoracic Duct (Thorotrast) in Cerebral Arteriography *Lancet* 1937 I 377
- PENFIELD, W "Epilepsy and Surgical Therapy" *Arch Neurol and Psychiat* 1936 XXXVI 449 Mechanism of Circumferential Contraction in the Brain *Brain* 1937 L 499
- PENNYBACKER, I and MEADOWS, S Normal Ventriculogram in Tumours of the Cerebral Hemisphere *Lancet* 1938 I 186
- SCHLESINGER, B Einführung in die Ventrikulographie 1937 Berlin Urban & Schwarzenburg
- TORKILDSEN, A and PIRHJELM Interpretation of Ventriculogram *Acta Neurol Scand* 1934 XXII 145
- TWINE, E W The Value of Radiology in Neurosurgery *Proc Roy Soc Med* 1936 XXI 1155

# CHAPTER IV

## INTRACRANIAL LESIONS

### INTRACRANIAL TUMOURS

AS IN other branches of radiology the diagnosis of intracranial tumours cannot be satisfactorily pursued by the radiologist without a working knowledge of the pathology and symptomatology of the lesions under review. It is not possible to give here more than a brief resume of the facts about intracranial tumours but a short sectional bibliography of works which furnish more information is provided at the end of the chapter.

Intracranial tumours occur with a frequency almost as common as that of tumours in any other part of the body. Their varieties are numerous and the analysis of a series of cases observed by one of us between the years 1927 and 1937 is shown in Table I.

TABLE I  
VARIETIES OF INTRACRANIAL TUMOURS (1927-1937 INCLUSIVE)  
(Benign and relatively benign types of tumour in italics)

	NO. OF CASES	PERCENTAGE INCIDENCY
<b>Glioma Group</b>		
Glioblastoma multiforme	144	19.5
Cerebral and pontine astrocytoma	67	8.4
<i>Cerebellar astrocytoma</i>	41	5.6
Medulloblastoma	26	3.5
<i>Oligodendroglioma</i>	26	3.5
<i>Ependymoma</i>	16	2.2
<i>Astroblastoma</i>	2	0.2
Ganglioneuroma	1	0.1
Pinealoma	2	0.2
<i>Glioblastoma polare</i>	3	0.4
Gliomatosis	1	0.1
Unclassified gliomas	23	3.0 (47%)
	347	
<i>Meningioma</i>	107	14.5
<i>Parasitary adenoma</i>	78	10.3
<i>Acoustic neurinoma</i>	44	6.0
<i>Congenital tumours (epidermoids, dermoid, cholesteatoma, etc.)</i>	35	4.7
<i>Blood vessel tumours</i>	34	4.6
<i>Cysts, benign (colloid cyst, etc.)</i>	9	1.2
Metastatic tumours	56	7.6
Sarcoma (primary)	3	0.4
Papilloma	1	0.1
Various benign	6	0.8
Various malignant	10	1.3
Tuberculoma	10	1.3
<b>Total</b>	<b>738</b>	

Approximately one-half of these tumours are benign ; the rest are malignant. The arrangement of the table is designed to separate the benign from the malignant, but the subdivision is not to be regarded as definitive : most varieties of the benign group occasionally exhibit malignant forms, while some in the malignant group are at times slow in their development and do not recur after operation. In Table II is set out a brief résumé of the radiological signs of intracranial tumours that were described in detail in previous chapters. This schema can be applied by the radiologist in the detailed consideration of any variety of intracranial tumour.

TABLE II

## SUMMARY OF THE RADIOLOGICAL SIGNS OF INTRACRANIAL TUMOURS

A *Plain X-ray Films.*

Direct radiological signs	<ul style="list-style-type: none"> <li>Calcification in tumour</li> <li>Thinning of bone over tumour with or without bulging, by pressure or by invasion</li> <li>Thickening (hyperostosis) of bone over tumour, by pressure or by invasion</li> </ul>
Indirect radiological signs	<ul style="list-style-type: none"> <li>Widening or narrowing of foramina</li> <li>Increased meningeal channels over and near tumour</li> <li>Displaced pineal shadow</li> <li>Increased convolutional markings</li> <li>Separated sutures (localised or generalised)</li> <li>Changes in sella turcica</li> <li>Enlargement of foramina of emissary veins</li> </ul>

B *Ventriculograms and Encephalograms (with various contrast media)*

Alterations in size, shape, and position of ventricular system, indicating the site of obstruction, or of deformation

C *Cystograms*—Contrast media in cysts connected with intracranial tumoursD *Arteriograms*—Tumours may produce.

- Displacement of part of arterial tree
- Dilatation or narrowing of a main cerebral artery
- Abnormal distribution of blood vessels.
- Aneurysmal dilatation of blood vessels
- Opaque shadows, from contrast medium in vessels of tumour
- Alterations in rate of circulation through intracranial blood vessels

*Early Symptoms of Intracranial Tumour*

It will be useful here to consider the more common symptoms that bring the patient under suspicion of suffering from an intracranial tumour. The first symptoms vary widely, depending on the situation of the tumour, its rate of growth, the pathological complications to which from its histogenesis it may be liable, and also on the type of the individual, especially as regards his personality and mental constitution. In each case, however, the clinical picture when viewed over a sufficient period of time is one of *progressive illness*, a picture of gradually or intermittently advancing disturbance of function. The progressive character of the symptomatology is what distinguishes intracranial tumour and allied conditions from other disorders of the brain.



**Hydrocephalic Symptoms** —The most common early hydrocephalic symptoms are headache vomiting papilloedema and drowsiness Others occurring as a rule at the later stage consist of paralysis of the cranial nerve especially the sixth pair epileptic attack extensor plantar responses and loss of deep reflexes

**Focal Symptoms** —Some tumours may produce symptoms of local disturbance of one part of the brain before there is any evidence of general rise of intracranial pressure for example the first symptom of a tumour in the vicinity of the right Rolandic fissure may be progressive disability of the left hand and this may occur long before the onset of headache or papilloedema But even when the focal origin of a symptom is clearly understood it does not always signify that the tumour is situated in the corresponding region of the brain since focal disorder may be produced at a distance from the tumour by distortion of the brain or by hydrocephalus The radiologist has seen from ventriculograms how one callosal gyrus may be jammed beneath the free edge of the falx cerebri by tumour at a distance and how the third ventricle may be distended in hydrocephalus from subtentorial tumour But the resulting symptoms are of relatively late onset focal symptoms that occur early in the course of an illness usually correspond to the site of the lesion

Among early focal symptoms of intracranial tumour must be considered the progressive loss of function in the cranial nerves the deafness which is usually the first sign of acoustic neurinoma and the visual disturbance in bitemporal hemianopia which are the first evidence of some sellar and suprasellar lesions

**Epileptic Attacks** —Minor or major epilepsy beginning in adult life without any history of head injury or hereditary taint is more often due to intracranial tumour than to any other cause and may precede by months or years the onset of other symptoms The epileptic attacks may be focal or general in character according to the situation of the tumour

**Progressive Dementia** —Mental symptoms are common as a late result of raised intracranial pressure but they occur also as the first symptom in a number of cases of supratentorial tumour especially in the form of failing attention and memory lack of initiative and deterioration of personality

**Disorder of Endocrine and Vegetative Functions** —These are common early symptoms of tumour of the pituitary and third ventricular regions and include disturbance of reproductive functions (amenorrhœa impotence etc) of growth (dwarfism gigantism etc) of metabolism (glycosuria obesity polyuria etc) and of sleep (hypersomnia insomnia)

**Cranial Swelling** —A visible or palpable swelling on the head may sometimes be the first evidence of an intracranial tumour and may be associated with localised headache independently of any alteration of intracranial pressure The radiological significance of localised cranial swellings has already been discussed (Chapter II page 37)

## PITUITARY TUMOURS

The pituitary gland is an endocrine gland, and its disorders produce profound effects on the rest of the body and on its functions. The majority of known lesions of the pituitary gland are expanding lesions which compress and stretch adjacent structures, thus producing signs of local disturbance in addition to the endocrine signs, of these the most important is loss of vision from pressure on the optic chiasm. The disturbance of vision is primarily bitemporal (or occasionally homonymous) hemianopia and thus often advances to complete blindness if the lesion is not treated. Hitherto endocrine treatment of pituitary disorders has not been attended with great success, and the best that can so far be achieved in most cases of pituitary disease is to preserve vision by relieving pressure on the optic nerves and chiasm. This is done by operations (which in the hands of experts have reached a remarkable degree of perfection), and also especially in acromegaly by X ray treatment.

In most cases the diagnosis of an expanding pituitary lesion should be made in the first instance by examination of the patient's visual fields, the X ray diagnosis being merely confirmatory, but in not a few instances radiographic examination demonstrates appearances from which not only the presence but also the pathological nature of the tumour can be inferred, and in some instances also it provides the surgeon with information as to how the tumour may best be approached at operation.

Pituitary tumours are almost invariably adenomas of the anterior lobe or epidermoid tumours of Rathke's pouch, the exceptions being rare cases of carcinoma. The growth of the adenomas is usually slow. In most cases it is for many years confined to the sella turcica, but eventually often only after ten or fifteen years, the diaphragma sellæ gives way and the tumour extends into the interpeduncular space or into the adjacent frontal or temporal lobe (Fig. 119).

Pituitary adenomas are of three kinds: (a) *chromophobe adenomas*, composed of chromophobe non granular cells; (b) *acidophil adenomas*, composed of cells whose granules stain with eosine and other acid dyes; this is the type of adenoma associated with acromegaly; (c) *basophil adenomas*, composed of cells whose granules stain with basic dyes.

**Basophil Adenoma** is small and does not produce any enlargement of the sella turcica or other change that can be seen in radiograms of the skull. It has recently been associated by Cushing with a syndrome of basophilism which is described in Chapter XL.

**Acidophil Adenoma. Acromegaly**—Acidophil adenoma produces acromegaly or, if the epiphyses are not united, acromegalic gigantism. This syndrome is too well known to require description here. From radiograms the radiologist can observe many of the changes that the disease produces: the generalised thickening of the bones and unevenness of their surfaces, the

tufting of the phalanges the great development of the lower jaw with consequent projection and separation from one another of the teeth, the prominence of the supraorbital ridges (due to the great size of the frontal sinuses) (Fig. 132) and in acromegalic giants kyphosis. In all bones the markings of muscular attachments are accentuated. Whenever there is an open epiphysis active bony growth is going on but the changes of acromegaly are not confined



FIG. 132. Acromegaly. The skilagram shows enlargement of the sella turcica and of the frontal sinuses, generalised thickening of the skull bones, and prolongation of the lower jaw. The semicircular shadow above the petrous bone is due to the cartilage of the external ear. (Case No. 135.)

to the skeleton. There is also generalised enlargement of the soft tissues and viscera (splanchnomegaly). Sexual dystrophy also occurs: males lose sexual desire and eventually become impotent, and in females amenorrhœa is often one of the earliest symptoms. Glycosuria and hyperglycæmia occur soon or later in about 25 per cent. of the cases.

The sella turcica is usually enlarged. As a rule the enlargement tends to be deeper than in other types of adenomatous enlargement: it is almost

deep as it is long often gourd shaped and the dorsum sellæ presents a scooped out appearance. There is much less destruction of bone by pressure than occurs in chromophobe adenoma, because in acromegaly the sella participates in the general bony overgrowth and is thus thicker and more resistant to pressure. This doubtless explains the severe headaches that often occur in acromegaly, in contrast to the absence of headaches in chromophobe adenoma which easily breaks through the thinned bony floor into the sphenoidal sinus. In a few cases of acromegaly the sella is identical with that seen in chromophobe pituitary adenoma, while in others its size is within normal limits.

**Chromophobe Adenoma**—This tumour is at least four times as common as the acromegalic adenoma, but its endocrine signs are much less conspicuous and the diagnosis is sometimes not made until the disease is far advanced and the patient almost blind. The presenting symptom is almost always failure of vision. In contrast to acromegaly, headache is inconspicuous. Endocrine signs may be completely absent, but many patients though they would pass for normal in the street show certain changes in habitus—a slight degree of adiposity, a pale delicate skin, a scantiness of hair on the trunk and face. As in acromegaly there is as a rule, sexual dystrophy.

In chromophobe adenoma the sella turcica is usually considerably enlarged, but in some cases its size may be within normal limits. The anterior lobe has an epithelial extension along the stalk of the pituitary gland from which supra diaphragmatic adenomas can arise. In these cases sellar enlargement may not occur, even though the tumour is large. The sella turcica may also be normal in size in the early stages of intrasellar adenomas. In this connection it must be noted that development of chromophobe adenomas may be extremely slow. For example, a patient with such a tumour may have amenorrhœa for ten or fifteen years before she experiences any symptoms due to compression of the optic nerves and chiasm. The radiological characteristics of the sellar enlargement are those which have already been described in Chapter II.

It is important to realise that the adenoma may arise in any part of the anterior lobe and that its effects upon the sella may be limited in the first instance to the neighbouring part of the sella. A tumour arising at the anterolateral aspect of the anterior lobe may produce thinning of the overlying clinoid process, or concavity of its under surface, while the other anterior clinoid process is still normal and the rest of the sella is only moderately enlarged. In other laterally placed tumours one side of the sella may become enlarged and deepened while the other side is unaffected, thus giving an appearance of a sella of double outline even in a perfectly lateral radiogram.

Calcification has been reported in chromophobe adenoma, and may also occur in the diaphragma sellæ over the tumour. In such cases the radiological appearances at times resemble those of cerebral aneurysm, and it may be necessary to perform arteriography to settle the diagnosis. In chromophobe

adenoma there are no radiological signs beyond those shown in films of the skull. The frontal sinuses are not enlarged.

In certain cases of epidermoid tumour of Rathke's pouch, where the tumour develops within the sella turcica rather than above it, and is not calcified, the radiological appearances may be identical with those found in chromophobe adenoma. Pituitary tumours occur in which there are slight signs of acromegaly combined with signs of chromophobe adenoma. The radiological appearances in such cases are the same as those of chromophobe adenoma.

### SELLAR AND SUPRASellar EPIDERMoid TUMOUR (Syn TUMOUR OF RATHKE'S POUCH, CRANIOPHARYNGIOMA)

Tumours of squamous epithelium are found within and above the sella turcica and they probably arise from remnants of Rathke's pouch, the evagination of the primitive buccal cavity from which the anterior pituitary lobe is developed. Remnants of this pouch are found in the pituitary gland in a large number of subjects and they can apparently start to grow at almost any age and may form large tumours. The tumours are benign. Sometimes they are composed of a solid mass of squamous epithelium, but at other times they contain one or more large cysts the fluid of which is of greenish hue and glistening from the presence of cholesterol crystals. Usually they are calcified sometimes intensely sometimes only slightly and in one part. The densest calcified tumours may be found to contain bone.

The tumour may be situated entirely within the sella turcica or may be entirely above it occupying the interpeduncular space and obliterating the third ventricle. In some cases the tumour is both within and above the sella. Cystic extensions of the tumour may burrow into the frontal or temporal lobes

or even into the parietal lobe (Fig. 119).

The symptoms produced vary greatly with the age of onset and with the situation of the tumour. In children growth is disturbed from compression of the anterior lobe of the pituitary gland and the Lorain type of dwarfism is produced. The dwarf is perfectly symmetrical and is of normal or even heightened intelligence, but his sexual development is in



FIG. 133.—Calcified suprasellar epidermoid tumour (Case No. 36).

abeyance. His epiphyses remain open even up to the age of 35, as in one case in our series. In adolescents the disturbance of growth may not be obvious by the time the other symptoms are acute. When the tumour does not begin until the patient is fully grown disturbance of habitus is not so profound but is a rule the skin becomes pale and delicate and there is fine wrinkling of the face and scanty growth of hair.

in appearance suggesting premature senility. Sexual dystrophy occurs from compression of the anterior lobe of the pituitary and the patient is often languid, lethargic and sleepy.

When the tumour extends or develops above the sella there may be disturbance of metabolism of carbohydrates and water from pressure on the hypothalamus. The patient may become fat usually only to a moderate degree and may suffer from excessive thirst and polyuria. The tumour often obliterates



FIG. 134.—Supra-sellar epidermoid tumour solid in its lower part cystic above. Case No. 177.

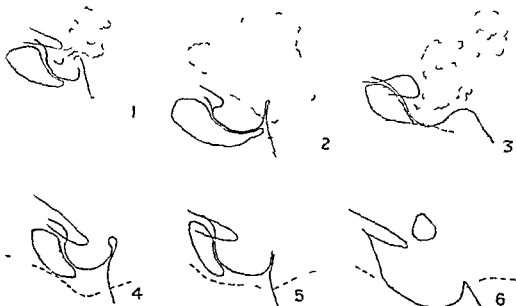


FIG. 135.—Six cases of epidermoid (Rathke pouch) tumour showing variations in the appearance of the sella turcica and of calcification in the tumour.

ates the third ventricle and blocks the foramina of Monro and the resulting hydrocephalus with its attendant headaches vomiting and papilloedema may be the most prominent feature of the clinical picture almost completely obscuring the manifestations of pituitary disorder in such cases from clinical examination the diagnosis of tumour of the posterior fossa is often suggested many neurologists confronted with such a case must have experienced a feeling of profound relief when the X rays revealed suprasellar calcification thus settling conclusively a diagnosis that would otherwise have been reached with difficulty As in pituitary tumours the optic chiasm is usually involved with resultant bitemporal or homonymous hemianopia but hemianopia is sometimes absent particularly in those cases in which the tumour arises in the floor of the third ventricle

There are great variations in the radiological appearances produced by epidermoid tumours depending mainly on the position of the tumour in relation to the sella turcica and on the presence or absence of calcification Calcification is the most conspicuous feature of these tumours and is usually diagnostic Sometimes it is widespread throughout the tumour as an area of great density (Fig 133) while at other times only a few flecks may be visible and then only in under exposed films diffusely scattered or limited to one part of the tumour Some tumours contain no calcium and cast no shadow at all as for example the large cystic and solid tumour shown in Fig 134

The appearance of the sella turcica varies When the tumour lies entirely above the sella the most common finding is partial or complete destruction of the dorsum sellæ and there is as a rule also diminished density of the rest of the sella The sella in such cases does not become enlarged often it is small and shallow (Fig 135) If there is no calcification the appearances presented in radiograms are not specific and we have several times seen exactly similar appearances in the sella produced by a glioma of the cerebral hemispheres at some distance from the sella When the tumour is entirely within it the sella enlarges in just the same way as in chromophobe pituitary adenoma and if there is no calcification it may be difficult to make a differential diagnosis from the radiograms However in epidermoid tumours there is often a tendency for anterior prolongation of the sellar enlargement beneath the anterior clinoid processes which are themselves sometimes unusually long In addition in non calcified intrasellar epidermoid tumour the clinical signs of hypopituitarism are usually much more intense than in chromophobe adenoma the skin of the face is more wrinkled pale and waxy the sexual dystrophy is more evident the extremities colder the blood pressure and basal metabolic rate lower

The ventriculographic appearances of epidermoid tumours are those of symmetrical hydrocephalus of the lateral ventricles often of severe degree with obliteration of the lower part or the whole of the shadow of the third ventricle (Fig 136) Cystic extensions of the tumour may be revealed radio

graphically by injecting air into them (Fig 119). Some times air may enter the cyst spontaneously (Fig 66). In encephalography the presence of an epidermoid cyst may obliterate the cisternæ chiasmatis and interpeduncularis and thus prevent their filling with air. While the fact that these cisterns have filled suggests that an epidermoid tumour is not present, this is not a reliable sign as we have seen them filled in the presence of such a cyst. Conversely the absence of good filling of the cisterns does not necessarily prove that there is an expanding lesion in the region of the tuber cinereum; it may result from errors in technique. Other X-ray signs of epidermoid cysts concern the bony abnormalities of dwarfism of the Lorum type (Chapter XL).

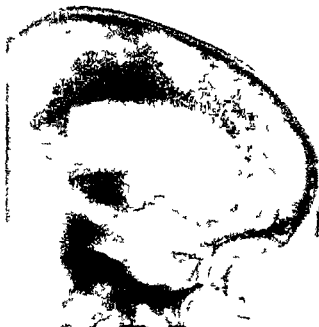


FIG 136 — Epidermoid (Rathke pouch) cyst of the third ventricle. The lateral ventricles are greatly dilated (though not the inferior horn). Only the upper part of the third ventricle is filled with air. Case R I 2397.

### MENINGIOMA

Meningiomas which comprise about 10 to 15 per cent of brain tumours, arise from the meningeal envelopes of the brain. Most of them are surface tumours attached to the dura, but some come from the falx cerebri or tentorium and a few are within the lateral ventricles and arise from the choroid plexuses, or from the membranes that pass to the surface through the great transverse fissure. For detailed description of the histological appearances the reader is referred to works on pathology (*Rio Hortega, Bailey and Bucy*). What is of radiological importance is that many meningiomas tend to form round masses of hyaline fibrous tissue known as psammoma bodies, and these sometimes become calcified. In a few cranial meningiomas bone formation also occurs. These changes may reveal part or the whole of the tumour in the radiograms. In a few cases meningiomas are multiple and are associated with bilateral acoustic tumours; in such cases psammomatous changes sometimes occur in the choroid plexuses of the lateral ventricles and render these structures visible in radiograms throughout their whole extent.



The superficial meningiomas have certain favoured sites alongside the sagittal sinus, over the Sylvian fissure, at the lesser wing of the sphenoid, in the olfactory groove and cribriform plate, and occasionally in the cerebello pontine angle. They are tumours of slow growth and are usually benign. Occasionally, however, they invade the overlying bone. They embed themselves in the brain as they grow, but do not invade it. The tumours are usually spherical, hemispherical or ovoidal and may attain an enormous size (up to 10 cm or more in diameter, and 300 gm or more in weight). Sometimes, however, the tumour grows *en plaque* and this type of meningioma usually measures only about 0.5 to 1 cm in its deepest part and is associated with excessive growth of the overlying bone.

Since meningiomas grow very slowly the neurological signs produced by them are often of an inconspicuous character and may be very difficult to interpret correctly, especially when they are of the so-called "false localising" type. For example a meningioma of one cerebral hemisphere may produce weakness of the same side of the body, and a meningioma in the frontal region may produce numbness and anaesthesia of one side of the face, a sign more suggestive of a tumour alongside the fifth nerve or Gasserian ganglion. The difficulty of localisation of meningiomas by clinical methods alone renders the radiological examination of great importance, especially as meningiomas usually give positive radiological signs that indicate not only the situation, but also the nature of the tumour. In certain situations however the clinical symptoms produced by meningiomas are fairly sharply defined. For full description of these the reader is referred to recent monographs on intracranial tumours, but the main points may be briefly mentioned.

**Parasagittal Meningioma**—The meningiomas alongside the sagittal sinus in the region of the Rolandic fissure commonly produce attacks of focal epilepsy, of a motor or sensory character in the contralateral limb. The contralateral limbs become progressively weaker, and when the tumour compresses the post-Rolandic region there is sensory loss of a cortical type (i.e. astereognosis, impairment of postural sensibility and of tactile discrimination, but no defect of appreciation of light touch and painful stimuli). In addition, there are usually the signs of rise of intracranial pressure—headache, vomiting, papillædema, drowsiness and slowing of mental processes.

Meningiomas alongside the sagittal sinus in the occipital region produce very few localising signs apart from an homonymous hemianopia which affects the lower quadrants before the upper, in some of them slight sensory loss of a cortical type may be observed in the contralateral limbs at times, from pressure on the parietal lobe. Parasagittal meningiomas in the frontal region anterior to the precentral gyrus may give no localising signs of a type that can be easily interpreted.

**Suprasellar Meningioma**—This tumour produces very slowly progressive disturbance of sight with primary optic atrophy and bitemporal hemianopia.

The sella turcica is seldom enlarged and usually there are no signs of endocrine disturbance such as occur with pituitary tumours. Hyperostosis or roughening of the tuberculum sellæ is the exception. On rare occasions a suprasellar meningioma is calcified.

**Olfactory Groove Meningioma**—In this tumour loss of smell in one or both nostrils is the most important clinical localising sign. In addition there is usually progressive loss of sight associated some times with bilateral papilloedema and at other times with primary optic atrophy of one eye and papilloedema of the other. As the tumour often extends backwards to stretch the optic chiasm bitemporal hemianopia is common. These tumours are also usually accompanied by rise of protein in the cerebrospinal fluid from the normal 0.02 to 0.04 per cent up to 0.1 per cent or even higher. Hyperostosis of the cribriform plate or of the bone between the cribriform plate and optic groove is a common but by no means invariable radiological sign. It is best seen in the true lateral projection as an elevation and thickening of the linear shadow which represents the floor of the anterior fossa behind the cribriform plate.



FIG. 13.—Hyperostosis on the lesser wing of the sphenoid bone due to meningioma. Postero-anterior and lateral projections. Case No. 1571.

**Sphenoidal ridge Meningioma**—This tumour rarely gives any obvious localising clinical signs apart from those associated with thickening of the orbit and exophthalmos, and involvement of the adjacent optic and oculomotor nerves. Hyperostosis of the bone can usually be seen radiologically. It varies from slight thickening of the base of the anterior clinoid process and the

adjacent inner part of the lesser wing of the sphenoid (Fig 137) to diffuse hyperostosis of the orbit (Fig 57) or of the middle fossa (Fig 110)

**Cerebello-pontine-angle Meningioma**—The symptoms of this tumour are the same as those of acoustic neuroma

### Radiological Signs of Meningioma

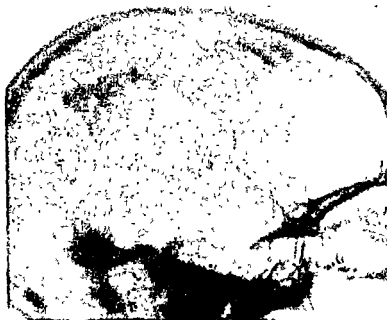
X ray examination gives positive localising signs in about 75 per cent of all cases of supratentorial surface meningiomas. These signs may be (1) changes in the overlying bone (2) changes in the cranial blood vessels (3) changes in the tumour itself and (4) arteriographic appearances

(1) **Changes in the Overlying Bone**—Changes in the bone over the surface of a meningioma may be produced by invasion of the tumour. In these cases the bone almost invariably becomes thickened forming a hard mass on the vault or temple (hyperostosis) (Figs 32 and 33) or if the meningioma is on the base of the skull producing proptosis of the corresponding eyeball by thickening of the bony walls of the orbit (Fig 57). The radiographic appearances of these bony changes usually show increase of density at the site of invasion (Fig 32) but sometimes in the vault the density of the invaded bone is less than normal (Fig 31)

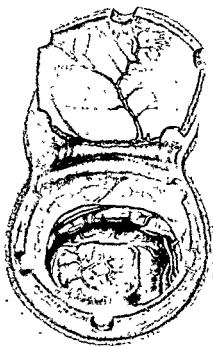
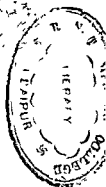
Changes may also occur in the bone overlying a meningioma without invasion of the bone by tumour. The most common of these is a small projection of the inner table of the skull (internal hyperostosis) at the centre of attachment of the tumour (Figs 40 140 and 159). This hyperostosis is best seen in tangential views. It may be produced by traction or pressure of the underlying tumour or by the establishment of fresh vascular connections between the dural attachment of the tumour and the overlying bone. In other cases large veins on the outer surface of the dura over the tumour may produce thinning and resorption of the overlying bone and even an inconspicuous nodular projection of the skull. Meningiomas which have spread outward through the dura but have not yet invaded the skull may produce a similar nodular projection

(2) **Changes in the Cranial Blood vessels**—Meningiomas of the falx tentorium and choroid plexus and a few of the surface meningiomas show no change in the vascular markings of the overlying bone. Superficial tumours of this type are usually avascular or get most of their blood supply from the cerebral rather than from the meningeal blood vessels. Most meningiomas are vascular tumours and their growth is associated with increase in size and number of the blood vessels of the overlying dura and bone. The increase of blood supply shows itself radiologically in the following ways

(a) *Increase in the size tortuosity and number of the meningeal channels on one side of the skull*. Enlargement of the main meningeal channel on one side in comparison with its fellow of the opposite side is a common finding in meningiomas of the convexity. Meningeal channels in an unusual place on one



(a)



(b)



FIG 138 (a) and (b)—A left parietal parasagittal meningeoma. In the operation sketch (b) the dura has been divided. On the inner aspect of the bone flap an enlarged meningeal channel is seen leading up to an internal hyperostosis. The pinpoint perforations in the hyperostosis are for fine vascular channels, which cannot be shown radiologically. The skigram (a) shows, however, numerous intramedullary vascular channels, especially in the region of the hyperostosis. The tortuosity of the vessel channels is well seen. Case No. 2851.

side of the skull should also raise a suspicion of the presence of a meningioma (Fig 138) When several meningeal channels on one side converge towards a point near the middle line it is practically certain that there is a meningioma beneath this point and efforts should be made to secure by a series of tangential views evidence of an internal hyperostosis at that point If a meningioma extends to each side of the middle line the meningeal channels on both sides may be enlarged

(b) *A cluster of fine branching or radiating channels in one area of the skull* is highly suggestive of meningioma In the same area there is usually seen in



FIG. 139 Calcified parasella meningioma Case No 6

some views one or more pin head holes which are due to blood vessels perforating the bone These channels can only be seen well in stereoscopic radio grams of high quality (Figs 40 and 138) The small arteries which produce them are most often on the inner surface of the skull but may be within the bone or on its outer surface

(c) *Increase in the size and number of diploic channels* may occur in the presence of a meningioma but it is difficult to make

a precise radiological diagnosis on such appearances They are not specific for meningioma and may occur with rise of intracranial pressure from any cause they may be as large on the contralateral side as on the side of the lesion It is necessary to add that where there is pathological increase of vascular markings it is not always possible to distinguish radiologically between meningeal channels and diploic channels

Evidence is accumulating to show that after removal of a meningioma there is fairly rapid diminution in the size of adjacent meningeal and diploic channels

(3) **Calcification**—In the tumour itself there may be diffuse calcification (Fig 139) or ossification (Fig 1) occasionally a portion only of the tumour may be calcified (Fig 140) and then it is difficult to distinguish the tumour radiologically from a glioma In some large meningiomas part of the edge of the tumour may be seen in profile as a faint but regular curved line of calcification and this appearance is distinctive

(4) **Arteriographic Appearances**—In arteriograms meningiomas may cast a diffuse but faint shadow but in some cases no shadow is visible and the only

signs are those of displacement of part of the cerebral arterial tree

### Differential Diagnosis of Meningioma

The following normal radiological appearances should be distinguished from those due to meningioma (a) bony or calcified plaques on the falx or elsewhere on the dura (Fig 2) (b) hyperostosis interna or diffuse hypertrophy of the inner table of the frontal bone (Fig 159) (c) normal diploic channels (Fig 38) Chronic leptomeningitis due to alcoholism or other cause may be associated with an excess

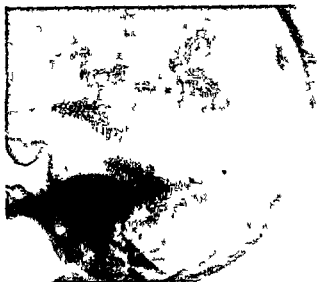


FIG 140—Internal hyperostosis with left occipital meningioma. Note the large vascular elements leading to the hyperostosis. The anterior margin of the tumour had a large calcification and shows a faint shadow indicated by an arrow. ← points to hyperostosis. Case No. 1344

sive proliferation of arachnoid granulations which lead to radiological changes strongly resembling meningioma (Fig 3). The cranial manifestations of local osteitis fibrosa (Fig 160a) may occasionally produce appearances resembling those of a basal meningioma with hyperostosis but in such a case the great and uniform increase of density of the bone and the wide extent of the changes will usually arouse suspicion and comprehensive radiographic examination of the long bones will rarely fail to reveal other manifestations of the disease.

In periosteal sarcoma of the skull the bony changes as seen radiologically are usually still slight when the tumour palpable on the surface of the head is already large (Fig 35) and this serves as a rule to distinguish the condition from meningioma with hyperostosis but in some cases of slow growing periosteal sarcoma and of metastatic carcinoma affecting the skull the radiographic appearances may be identical with those of a meningiomatous hyperostosis and only histological examination will settle the diagnosis. This is not surprising, since the reaction of the skull to invasion by tumour does not appear to be appreciably influenced by the histogenesis of the invading tumour.

### GLIOMA

Gliomas comprise nearly 50 per cent of all intracranial tumours. They are tumours of neuroglia, the supporting epiblastic connective tissue of the brain, and they present a great variety of widely differing clinical and histo-

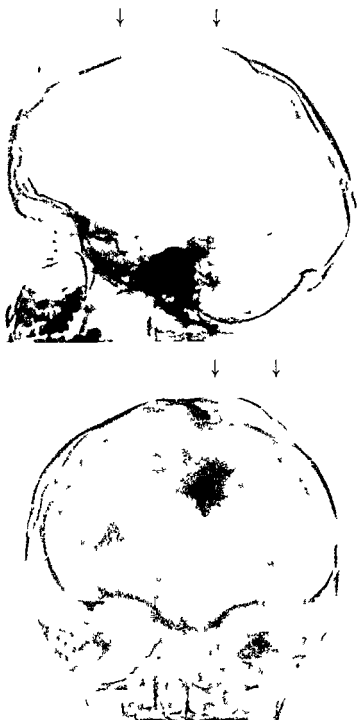


FIG. 141.—Bulging and thinning of the right parietal bone in a case of astrocytoma of the right frontal and parietal lobes in a child 15½ years. Case No. 933.

logical pictures. Some gliomas are extremely malignant and these are composed for the most part of poorly differentiated glial cells. With these tumours are grouped some that are probably neuroblastic in nature. It will be recalled that neuroglia and nerve cells have a common origin from the embryonic medullary epithelium. Other gliomas are benign and are composed of well differentiated glial cells. There are all gradations between the very benign and very malignant glioma. The commoner varieties include the following: (1) glioblastoma multiforme (spongioblastoma multiforme) (2) medulloblastoma (3) astrocytoma and astroblastoma (4) oligodendroglioma (5) ependymoma. For full description of the histological types the reader is referred to the works on the pathology of intracranial tumours.

The direct radiological signs of gliomas are calcification and thinning and (in young subjects) bulging of the skull over the tumour. Calcification occurs in about 20 per cent of gliomas. It may be scattered diffusely or limited to one small area of the tumour and the intensity of the process as indicated by the density of the shadow seen in radiograms varies greatly. The distribution of the calcification usually gives a characteristic radiological pattern in oligodendroglioma and sometimes in astrocytoma but in many cases it does not indicate the pathological type of the glioma (Figs 63 and 142). Localised thinning of the skull occurs over slow growing surface gliomas especially in children when it may be accompanied by localised projection (Fig 141).

**Glioblastoma Multiforme (Spongioblastoma Multiforme)**—This is a rapidly growing malignant tumour found most commonly in the white matter of the cerebral hemisphere in middle aged people. To the naked eye it is often necrotic and hæmorrhagic and cysts single or multiple are common. It usually recurs rapidly after removal. In a few cases it is radio sensitive. The clinical picture depends on the situation of the tumour but a fairly rapid course and early onset of stupor are common features. Direct radiological signs are not common in glioblastoma multiforme. In some of them a small portion of the tumour may be calcified but generalised calcification does not occur. The probable explanation of such calcification is that the tumour arises from dedifferentiation or reversion of a slow growing calcified focus of abnormal glial elements.

**Medulloblastoma**—This is a tumour of the cerebellum usually in childhood. It grows rapidly and tends to spread through the nervous system along the cerebro-spinal pathways. It is very radio sensitive so that successive recurrences of symptoms can be dealt with by X ray treatment but eventually after a period of one to five years it fails to respond to further treatment. Calcification in this tumour is an event of great rarity and the radiological signs are usually limited to those of raised intracranial pressure.

**Astrocytoma and Astroblastoma**—In the cerebellum astrocytoma occurs as a well differentiated benign tumour and in the cerebral hemispheres as a less well defined tumour that is not as a rule histologically homogeneous and is in fact often mixed with astroblastic or glioblastic structures. Cyst



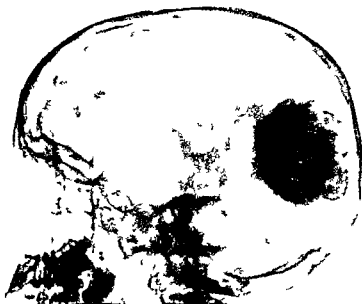


FIG. 142—Calcified oligodendroglioma of the right occipital lobe in a girl aged 12 years.  
Case No. 1733

formation is common in all varieties of astrocytoma. Calcification is rare in cerebellar astrocytoma but occurs with considerable frequency in cerebral



FIG. 143—Oligodendroglioma of the left callosal gyrus and adjacent parts. Case No. 24

astrocytoma, when it may be diffusely scattered through the whole tumour or localised to one part of it. The density of the calcification varies from case to case, as does its shape. Cerebral astrocytoma is the most common variety of tumour to produce localised thinning and projection of the overlying skull.

**Oligodendroglioma**—This tumour is found in the cerebral hemispheres of adults. It is composed of small round oligodendroglia cells, mixed with astrocytes. The tumour is of relatively slow growth, but is liable to sudden hæmorrhages, at times fatal. Calcification is common in oligodendroglioma, and frequently has a characteristic distribution in wavy lines along individual convolutions (Fig. 143). In its spread the oligodendroglioma frequently invades the brain without disturbing its convolutional pattern.

**Ependymoma**—This is a tumour that occurs in the walls of the ventricles, especially alongside the fourth ventricle, and also in the white matter of the cerebral hemisphere, doubtless arising there from rests of ependymal cells, such as are not infrequently found in the brain at some distance from the ventricular wall. The tumour is one of slow growth, which does not give rise to severe symptoms as a rule until it has attained a large size. This is especially the case in ependymomas of the cerebral hemisphere of young children. These may weigh as much as 400 gm. and they are usually calcified. In posterior fossa ependymomas calcification is rare. Indeed, in all tumours of the cerebellum calcification is so rare that it is not of value for differential diagnosis.

### ACOUSTIC NEURINOMA

The acoustic neurinoma is a benign tumour arising usually from the vestibular portion of the eighth nerve in its course through the cerebello-pontine angle from the porus acusticus to the pons. Occasionally the tumour arises on the nerve within the petrous bone. It may be bilateral and is then associated with von Recklinghausen's neurofibromatosis. The tumour is quite benign, and produces its harmful effects by pressure on the adjacent pons, cranial nerves and cerebellum.

The clinical picture is one of progressive unilateral cerebellar disorder, with deafness on the corresponding side, and to a lesser degree, involvement of the sixth, seventh, and other adjacent cranial nerves. With this are associated the signs of raised intracranial pressure. The protein content of the cerebrospinal fluid is almost invariably raised.

Radiological proof of the presence of an acoustic tumour is rarely called for, since the clinical picture of the condition is so sharply defined. This is fortunate, for, apart from the very rare occurrence of calcification in the tumour, the only direct radiological sign is dilatation of the porus acusticus, and that is far from constant, even though most acoustic tumours occupy the porus. Furthermore, according to Mayer, slight differences in the width of the porus

on the two sides may be within normal limits. In a few cases there is also diminished density of the apex of the petrous bone (Fig 59). By ventriculography it can be shown that these tumours produce in addition to internal hydrocephalus narrowing of the aqueduct of Sylvius and displacement of it to the opposite side.

### METASTATIC TUMOURS

Metastatic tumours constitute 7 to 8 per cent of all intracranial tumours. The most common primary tumour is carcinoma of the hilum of the lung but other frequent primary tumours are carcinoma of the breast, prostate, kidney (Grawitz type) and melanotic carcinoma of the skin or eye. In a considerable number of cases the first symptoms of illness are produced by the cerebral metastases and this fact should always be borne in mind during clinical investigation of cases of intracranial tumour in which the illness is of short duration.

Apart from the rare occurrence of metastatic osteogenic sarcoma of which an example was recently reported by Courville, metastatic tumours are not calcified and the chief importance of simple radiography in these cases is to reveal clinically silent primary carcinomas of the lung. Ventriculography may sometimes arouse suspicion that a case under investigation is actually one of metastatic tumour when in a patient with a short history of illness and obvious ventriculographic evidence of a tumour in one cerebral hemisphere there is also dilatation of both lateral ventricles. In rapidly growing glioma of one cerebral hemisphere the ventricle on the side of the lesion is usually collapsed. In rapidly growing metastatic tumour of one cerebral hemisphere the corresponding lateral ventricle is usually also collapsed if the tumour is solitary but if as is not uncommon, there is a further metastatic tumour in the cerebellar vermis then the ventriculographic appearances are those of a hemisphere tumour plus internal hydrocephalus. Similar appearances may be seen in ventriculograms of hemisphere gliomas after an illness of a year or more when the glioma has extended into the midbrain and has blocked the aqueduct of Sylvius but in cases with a short history this ventriculographic finding is more commonly produced by metastatic tumour than by any other cause.

Metastatic tumours may develop in the dura mater or overlying skull and may then grow quite slowly and produce changes in the bone as seen by X rays (Fig 165).

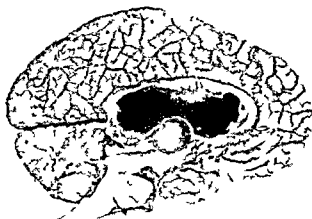
### COLLOID CYST OF THE THIRD VENTRICLE

Colloid cyst of the third ventricle is a small spherical cyst usually between 1.5 and 2.5 cm in diameter arising from the anterior part of the roof of the third ventricle at the level of the foramina of Monro (Fig 144a). It has a thin

wall, lined by a single layer of columnar epithelium and it contains thick gelatinous fluid. Situated as it is in the third ventricle at the exit of each lateral ventricle it commonly produces internal hydrocephalus usually in the form of intermittent attacks of great and even fatal intensity.

The clinical signs of this lesion are not often distinctive. Simple roentgenograms may show slight enlargement of the sella turcica and other signs of hydrocephalus but are not of localising value. Certain diagnosis of the site of obstruction in these cases of colloid cyst can only be made by ventriculography after complete replacement of the ventricular fluid by air and employing the special projection to display the anterior part of the third ventricle. The cyst is usually sharply outlined as a filling defect in the upper anterior part of the third ventricle (Fig. 144b). But sometimes there is complete blockage of the foramen of Monro and no air in the third ventricle. Colloid cyst can be suc-

cessfully removed from the third ventricle by an approach through the dilated anterior horn of the lateral ventricle and the foramen of Monro. The diagnosis depends mainly upon ventriculography.



(a)



(b)

FIG. 144. (a) Lithol image of specimen showing a colloid cyst filling part of the third ventricle. The cyst projects up into the floor of the lateral ventricle and downwards into the third ventricle.

(b) Ventriculogram of the patient. The dotted line shows the outline of the third ventricle. Its anterior end is not greatly dilated when compared with the degree of dilatation of the lateral ventricle. (Case No. 1875).

### CHOLESTEATOMA AND ALLIED TUMOURS

*Cholesteatoma* is an epithelial tumour. It has a thin wall lined by squamous epithelium and an interior of fat and cholesterol crystals the products of desquamated epithelium. These tumours occur in the brain at various sites above the sella turcica, in the third and lateral ventricles in the pineal region on the surface of the cerebellar vermis and on the surface of the cerebral hemisphere. In similar situations there occur *dermoid tumours* which differ only from cholesteatomas in containing hair and having a lining membrane

which is the homologue of true skin rather than of simple epidermis (Fig 145).

*Teratomas* containing more highly organised structures derivatives of all three germinal layers are also found rarely within the cranium (Fig 64).

All these tumours must be regarded as arising from rests of primitive cells that have become displaced during development of the embryo. They are not to be confused with the cholesteatomatous masses similar in structure to intracranial cholesteatoma which occur in the mastoid



FIG. 145.—Hurry dermoid at the foramen of Magen lie  
Case No. 1586

process after chronic suppuration. These masses probably arise by epithelial ingrowth from the auditory canal though the fact that they are sometimes found in patients with an intact ear drum and no history of otitis does suggest that some of the petrous cholesteatomas are truly neoplastic. Under the name of intratemporal epidermoids Jefferson and Smalley have described six cases in which the presenting symptoms were a slowly progressive facial palsy and deafness without otitis. The condition is only recognised with certainty by radiograms of the petrous bone especially the fronto occipital projections which show cyst like spaces with a capsule of condensed bone. These areas of erosion may even extend into the medial part of the petrous bone. Neoplastic cholesteatomas may be found also in the diploe of the skull where they produce great thinning of the skull.

Intracranial cholesteatomas rarely give rise to direct radiographic signs though calcification has been reported as occurring in them. In most situations their ventriculographic appearances are not distinctive but some large



FIG. 146.—Antero-posterior and lateral encephalogram of a large left fronto-parietal cholesteroloma. In the lateral projection the tumour is outlined by a congeries of air-filled sulci (see No. 20).

cholesteatomas of the cerebral hemispheres produce air pictures that are unlike those of other tumours. Fig. 146 shows the encephalogram of such a case. The tumour was a large cholesteatoma of the left parietal region presenting on the surface of the hemisphere and embedding itself deeply in the subjacent brain. The radiographic appearances show remarkably little lateral displacement of the ventricles and in the position of the tumour there are numerous interlocking air filled channels giving a mottled appearance. It is evident that the tumour is surrounded by cerebrospinal fluid channels which are in free communication with the lateral ventricle and possibly also with the subarachnoid channels on the surface of the brain. The complete absence of headache and papilloedema in this case is doubtless explained by the singular lack of obstruction to the flow of cerebrospinal fluid. Pattison has reported a similar case.

### CHORDOMA

Chordoma is a rare tumour which arises from remnants of the upper end of the notochord on the clivus below the dorsum sellæ. It usually destroys the basisphenoid and sometimes produces enlargement of the sella turcica indistinguishable from that of a large pituitary tumour. It is not calcified. Clinically the picture may resemble that of pituitary tumour or of pontine tumour. Multiple cranial nerve palsies are always present.

### PAPILLOMA

Papilloma is a rare tumour which arises from the epithelial covering of the choroid plexus of the lateral third or fourth ventricles. The tumour expands within the ventricle and raises the protein content of the cerebrospinal fluid and since it occupies a pre-existing space it may attain considerable size without producing headache, papilloedema or other signs of raised intracranial pressure. The tumour is not calcified. The ventriculographic signs of papilloma of the lateral ventricle like those of other intraventricular tumours may differ considerably from those of most tumours of the cerebral hemisphere inasmuch as the ventricle on the side of the lesion is seen to be dilated instead of being collapsed. When this dilatation is due to tumour rather than to obstruction of part of the ventricle by a more proximally situated tumour the air shadow is fainter than that of the normal ventricle or that of a ventricle dilated as a result of gliosis or other contracting lesions of the brain. Papilloma is sometimes found in the substance of the cerebral hemisphere quite unconnected with the choroid plexus of the lateral ventricle. Such tumours doubtless arise from embryonic rests of choroid plexus epithelium in the white matter. They may be associated with cysts which are shown by ventriculography to be diverticula of the lateral ventricles.

## TUBERCULOMA

Infection of the brain with tubercle bacilli from some distant focus may result in the formation of one or more discrete masses of granulomatous material known as tuberculoma. The size of the lesion varies between 1 cm and 6 cm in diameter. The common sites are in the cerebellum, centrum semi-ovale and midbrain. Clinically, tuberculoma usually behaves like an intracranial tumour but in some cases the lesion has an inconspicuous development, becomes quiescent, and the presenting symptom is one of epilepsy unattended by any signs of raised intracranial pressure or other symptoms of a progressive lesion. To the naked eye at operation the lesion may resemble a glioma but in most cases a past history of tuberculous infection in other parts of the body is obtainable which should put the surgeon on his guard. The importance of this is that the surgical treatment should be either radical excision of the tuberculoma or simple decompression, biopsy or partial removal of the mass is almost always followed by fatal tuberculous meningitis.

In chronic cases, which are however, relatively uncommon tuberculoma may be calcified (Fig 147). The shadow may be lobulated irregular, or circular. The margins are sharply defined and may present a crenated outline. Calcified tuberculoma may exist without ventricular deformation.



FIG 147—Multiple calcified tuberculoma in the brain (Note the slightly crenated outline of the shadows). This case is unverified. The patient, a male aged 18, had suffered from epilepsy for four years. There was a strong family history of tuberculosis and the patient had had pneumonia at the age of 2 during which he had a fit. Cephalography showed normal ventricles. Case No 2191.

## PARASITIC CYSTS

Cysticercosis in man is caused by ingestion of the eggs of the tapeworm, *Tænia solium*, and the consequent invasion of the body by the embryos. The embryos may pass to any part, but most commonly to the subcutaneous tissues, muscles, and the grey matter of the brain, where they develop into cysticerci,



the larval stage of the parasite. The larva may exist as a tiny nodule for months or years but then becomes cystic and swells. According to *MacArthur* this change is associated with the death of the larva. As the parasite degenerates it may become calcified.

Because of their predilection for the brain cysticerci produce epilepsy and this is the main almost the only symptom of the disease. Cysticercosis is



FIG. 148.—Cysticercosis. Scattered calcified cysts in the brain. Case No. 30 J.

most commonly seen in this country in those who have resided in India and the onset of epilepsy in anyone who has resided in India raises strong suspicion of cysticercosis. Proof of the disease rests on the demonstration of the parasite in an excised subcutaneous nodule or in the discovery by X rays of calcified cysticerci in the muscles of the limbs or in the brain (Fig. 148). The different

radiological appearances presented by cysticerci have been described in detail by *Morison*.

**Hydatid Disease** is the larval stage of *tænia echinococcus* and is found sometimes in the brain though more commonly in the liver, lungs and other organs. It is rare in the British Isles but common in Australia. In the brain there may be one or more cysts. Unlike cysticercus the hydatid cyst attains a large size and it behaves like an intracranial tumour. It is usually fatal before it undergoes calcification but it may produce in addition to signs of raised intracranial pressure distortion and great thinning of the skull over the cyst especially when the cyst is extradural as in a case reported by *Kneebone*. According to *Deu* the very large solitary cerebral cysts are found almost exclusively in children.

### INTRACRANIAL ANEURYSM

Most intracranial aneurysms occur on the base of the brain in the circle of Willis or nearby in the arteries connected with it. While some are due to syphilis and others to arrest of infective emboli in the cerebral arteries (mycotic aneurysm) most intracranial aneurysms result from congenital

weakness in the muscular coat of the artery at one point, usually at or near the origin of a branch (*Turnbull*)

The majority of intracranial aneurysms produce no radiological signs, and in the past there have not been many cases in which radiology has contributed to the diagnosis. Nevertheless, in a few cases the sac of the aneurysm contains calcium in its wall (Fig 149), in others the aneurysm may erode the adjacent bone and thus provide indirect radiological evidence of its presence. In other cases signs of pressure on the optic chiasm, optic tracts, or optic nerves may be associated with enlargement of the sella turcica and signs of dyspituitarism, and a diagnosis of pituitary tumour is made. These cases occur with sufficient frequency to be of interest to the radiologist, and it is possible that with increasing experience he may learn to suspect the true nature of the lesion from the manner in which the sella and adjacent bony structures are deformed. The enlargement of the sella resembles closely that caused by pituitary adenoma, though in aneurysm there is a greater likelihood of destruction of the corresponding anterior clinoid process. *Jefferson* has pointed out that some intracranial aneurysms cause erosion of the lower and outer wall of the optic canal and enlargement of the sphenoidal fissure.



FIG 149—Calcification in the wall of a small aneurysm of the posterior communicating artery. Case No 2206

The development of cerebral angiography renders it likely that many more intracranial aneurysms will be demonstrated radiologically in the future, and it is thus important that radiologists should be conversant with the clinical picture produced by intracranial aneurysms. For details of the symptoms and signs of intracranial aneurysms the reader is referred to the papers of *Fearnside*, *Symonds*, *Allbright*, and *Natthass*. Some aneurysms give rise to no symptoms and are only found at necropsy. On the other hand, symptoms may begin at any age and are of two main groups: (1) symptoms due to rupture of the aneurysm and hæmorrhage, (2) symptoms due to pressure of the aneurysm on surrounding structures.

(1) When an aneurysm ruptures it bleeds usually into the basal cisterns of the subarachnoid space, for most intracranial aneurysms are situated there. In a few aneurysms of the internal carotid artery the hæmorrhage may be subdural, and when the aneurysm is of the anterior cerebral artery the hæmorrhage sometimes extends into the substance of the brain and even into the lateral ventricle. The hæmorrhage may be so severe as to produce sudden death. More often there is temporary unconsciousness, followed by violent headache extending from one side of the forehead to the back of the neck.

The clinical picture then closely resembles that of purulent meningitis but can be distinguished from it by lumbar puncture which shows free blood or its products in the cerebrospinal fluid. In many of these cases large retinal hæmorrhages appear around the optic discs (*Riddoch and Goulden*). Many patients recover from one or more attacks of this nature.

(2) *The local signs of aneurysm* may occur during an attack of hæmorrhage or they may be produced by enlargement of the aneurysm without rupture. The most common local signs are those due to interference with the third cranial nerve but the second, fourth and sixth cranial nerves and the ophthalmic divisions of the trigeminal nerve of the same side may also be involved. The optic chiasm or the optic tract of the same side may also be pressed upon. Ipsilateral proptosis may occur. The sella turcica may become enlarged and in part destroyed. When there are no episodes due to subarachnoid hæmorrhage these local signs may suggest the presence of tumour such as meningioma of the lesser wing of the sphenoid and arteriography may then be of great importance in diagnosis. Cerebral aneurysms as a rule do not produce an audible bruit.

In recent years ligation of the carotid artery has been employed for some congenital cerebral aneurysms with promising results (*Bramwell and Dott*).

### ANGEIOMATOUS MALFORMATIONS

The cerebral blood vessels give rise to aneurysms which have already been described to angiomatic malformations and to tumours (cavernous and capillary hæmangioma and hæmangioblastoma). The tumours do not present any direct radiological signs but the angiomatic malformations are of considerable radiological importance. They consist of coiled masses of blood vessels on the surface or in the substance of the brain. These vessels may consist solely of veins (venous angioma) or of a mixture of arteries and veins (arterio venous aneurysm, angioma racemosum arteriale).

**Venous Angioma**—Venous angioma consists of abnormal collections of veins on the surface of the brain sometimes extending also deeply into its substance. The veins may be small and coiled or they may be very large. *Their histological structure is abnormal and their walls are often calcified* (Fig 150a). In some cases the adjacent brain cortex is calcified. They have been found in the cerebellum but are much more common in the cerebral hemispheres. They are usually unilateral and when the angioma is wide spread the underlying cerebral hemisphere is abnormally small. Similar abnormal collections of vessels may be found on the meninges. The condition may be familial.

The main symptom of venous angioma of the brain is epilepsy. Sometimes this does not come on until adult life and then the mentality of the patient is usually unaffected. Most venous angiomas give symptoms in

childhood, including epilepsy and some degree of mental deficiency. Often there is also a nevus, or port-wine stain, on the face usually on the same side as the cerebral angioma (Fig 150*b*). The corresponding eye may show buphthalmos, congenital glaucoma, coloboma, or other developmental abnormality. The neurological signs and to a certain extent the type of the fits depend on the situation of the angioma. Most often the angioma covers the greater part of one cerebral hemisphere and the fits affect chiefly the opposite arm and leg, which are weak and spastic. Sometimes however the angioma is practically confined to the occipital lobe and homonymous hemianopia is the only sign. Papilloedema and other signs of rise of intracranial pressure do not occur.



FIG. 150—(a) Calcified angiomatous malformation of the left occipital lobe together with hemiatrophy of the left side of the cranium in a patient with left frontal nevus. The Sturge Weber syndrome. Case No. 2925.

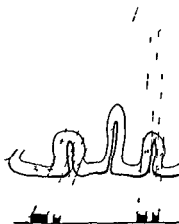
Treatment of these cases is unsatisfactory. Operation serves no useful purpose and hitherto radiation treatment has not been successful.

Many venous angiomas give no radiological signs but others are calcified, and in some there is a very distinctive type of radiological appearance consisting of tortuous, parallel double lines of calcification (Fig 151*a*). This type of calcification occurs in some though not in all cases of venous angioma that are associated with facial nevus. On superficial examination of the radiogram the calcification appears to be in the walls of the abnormal cortical blood-vessels, but histological studies have shown that it is actually in the shrunk convolutions of the angiomatous area. Closer inspection shows that the calcification is not limited to the parallel lines, but exists in varying degrees of density, in the surrounding cortex. *Bergstrand* has explained the lines visible



(a)

FIG 151 (a) — Angiomatous malformation of the right occipital lobe in a man aged 13½ who suffered from fits preceded by visual hallucinations. Case No 556



(b)

FIG 151 (b) — Diagram to explain the appearance of double lines of calcification thrown by the calcified cortex of the Sturge Weber Syndrome. (After Bergstrand)

in radiograms as corresponding to those parts of the cortex that are end-on to the direction of the rays (Fig 151b)

**Arterio venous Aneurysm** (*syn* arterial or racemose angioma) — Arterio venous aneurysm consists of a coiled mass of vessels, some of them arteries, some of them veins, on the surface of the brain and extending into its substance (Fig 152). The essential feature of the lesion is that there is a direct communication between the abnormal arteries and veins as a result of an error of development of the vessels. At operation a stream of bright red blood can be seen pouring into the huge veins. The veins become more and more dilated and may rupture from time to time giving rise to sudden apoplectic attacks. The main artery of supply to the lesion is usually a branch of the circle of Willis. becomes tortuous and dilated. The whole lesion increases in size and acts like an intracranial tumour in producing rise of intracranial pressure. Arterio venous aneurysms are found usually on

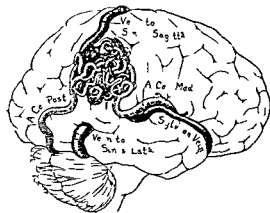


FIG 152 — Arterio venous angiomatous malformation. (From Cushing and Bailey)

the cerebral hemispheres, sometimes diffuse, but more often localised to the distribution of one cerebral artery, but they also occur on the cerebellum, when the vertebral artery is usually the artery of supply. The walls of the blood vessels are subject to an unusual degree of degeneration, and calcification of the adventitia and media are not uncommon, this has occasionally been observed radiologically. The shape of shadow rarely suggests a vascular pattern. It is spherical or linear and may resemble the shadows of gliomas.

Although congenital in origin these lesions usually do not give rise to symptoms until adult life, and the clinical picture is quite different from that of venous angioma. It is true that epilepsy is an almost invariable symptom when the lesion is supratentorial, but there are, in addition, progressive focal symptoms corresponding to the situation of the lesion, and signs of raised intracranial pressure—papilloedema, headaches, and so forth. Sometimes the scalp is unusually vascular, and always there is a pronounced systolic bruit over the arterio venous aneurysm. It is usually only by detecting the bruit on auscultation of the head that the nature of the lesion is suspected before operation, though sometimes there are vascular abnormalities of the overlying skull (Fig 39). The murmur is maximal over the site of the lesion, but is usually conducted to other parts of the cranium and is often very loud. It may be difficult to hear these murmurs over the hairy part of the scalp until the scalp is shaved.<sup>1</sup>

The treatment of arterio venous aneurysm of the brain is much more satisfactory than that of venous angioma. The aneurysm cannot be excised without very grave risk of fatal hæmorrhage, or of subsequent loss of function from severe damage to the brain, but decompressive operations are very satisfactory in relieving intracranial pressure, and in suitable cases ligation or narrowing of the appropriate internal carotid is also of service. There are cases in which the bruit is arrested by compression of the carotid artery of the corresponding side, and in which the aneurysm cannot be filled by arteriography of the carotid of the opposite side. Cushing has shown that X ray treatment may be effective in causing the aneurysm to become thrombosed, as evidenced by cessation of the bruit and relief from symptoms.

The arteriograms of arterio venous aneurysm vary considerably from one case to another but always show some departure from the normal vascular tree. The internal carotid artery is often dilated. In some part of its dis-

<sup>1</sup> A similar murmur is also heard in cases of traumatic carotico-cavernous arterio venous aneurysm—a lesion that is not considered here, since it gives no radiological signs. Cirsoid (arterio venous) aneurysm of the scalp also gives a loud systolic murmur. It should be noted also that a cranial bruit is not pathognomonic of arterio venous aneurysm for it can be heard in many normal infants and in many children and young adults who suffer from intracranial tumour with great rise of intracranial pressure. It is then usually heard best over the lateral and sagittal sinuses. A systolic murmur is also heard sometimes in adults over a meningioma, when the bone and dura overlying the tumour are very vascular but this murmur is rarely intense.

tribution there are tortuous branches excessive both in size and number. These vessels may lead into large areas of irregular shape and uneven density which are filled with thorotrast and large venous channels may be seen leading from the lakes of thorotrast (Fig. 131).

### INTRACRANIAL HÆMORRHAGE SUBDURAL HÆMATOMA

Intracranial hæmorrhage is caused by diverse lesions of blood vessels such as aneurysms and angiomatic malformations, atheroma, injury, and also by



FIG. 123. Ventriculogram of left subdural hæmatoma showing displacement of the ventricular system to the right and a large collection of air around the subdural clot.

hæmorrhage from the vessels of a tumour. The bleeding may take place in the subarachnoid or subdural space into the substance of the brain or into the ventricular system. Radiology plays little part in the diagnosis of these cases except under the conditions that a massive clot forms so slowly as not to kill the patient. Sublethal clots are usually found in the subdural space (*subdural hæmatoma*), but may at times occur in the substance of the brain.

In the brain these clots usually become completely absorbed with shrinkage of the brain and formation of a scar and traction diverticulum of the ventricle (Fig. 125). Occasionally, during absorption of the clot there may be also deposition of calcium salts. We have seen this at necropsy but not radiologically.

The radiological appearances of subdural hæmatoma after air injection are distinctive. This condition may follow acutely upon a severe head injury but more commonly it develops insidiously through a period of weeks after a relatively slight blow on the head. In the acute cases the patient soon becomes comatose but when the clot develops slowly the clinical picture is one of headaches and intermittent drowsiness through a period of weeks. In spite of the presence of a massive clot compressing one or on occasions both cerebral

hemispheres there is little or no weakness of the limbs. The symptoms and signs may be so ill defined that diagnosis is difficult without air injection. If ventriculography is undertaken the clot may be encountered when the dura is opened and the diagnosis is thus settled without air injection. But as subdural clots occur more over the fronto parietal region than in the occipital region and are moreover enclosed in a membrane it sometimes happens that they are not found through the burr holes and ventriculogram is accordingly performed. The ventricles are small and are displaced from the clot which is usually though not always unilateral. The appearance of the ventricle on the side of the clot is not distinctive of a lesion in any one portion of the corresponding hemisphere and the true nature of the trouble would remain obscure if it were not for the fact that a large amount of air collects in the subdural space around the clot and shows clearly in radiograms (Fig 153).

Similar appearances of subdural air are found in subdural hematoma after encephalography. It is a remarkable fact that a subdural clot which can grossly compress the brain is yet surrounded by an area of low pressure into which air will pass during ventriculography or encephalography.

### ABSCESS OF THE BRAIN

The main causes of abscess of the brain are infections of the temporal bone infections of the frontal and other accessory nasal sinuses bronchiectasis and empyema foreign bodies within the cranium focal osteomyelitis of the skull, and staphylococcal pyæmia from boils carbuncles etc. Abscess of the brain may be acute severe cerebral symptoms supervening within a few weeks of the initial intracranial infection or it may be chronic lasting for as long as twenty years before giving rise to severe symptoms and then producing a clinical picture closely resembling that of intracranial tumour.

The value of simple radiography in brain abscess is limited because it is uncommon to find radiographic signs. On rare occasions there is enough gas in the abscess produced by anaerobic organisms to show in radiograms and this provides valuable evidence of the precise situation of the abscess. The presence of gas producing organisms does not indicate a virulent infection of the brain. Occasionally also the wall of a very chronic brain abscess may become calcified.

While direct radiological demonstration of the presence of a brain abscess is an uncommon event yet in chronic abscess indirect signs of raised intracranial pressure are often present. X-ray examination will also commonly demonstrate the cause of brain abscess such as a diseased frontal sinus and this may be of importance in individual cases in arriving at a successful diagnosis. Ventriculography is often of use in the diagnosis both of acute and of chronic abscess.



## HYDROCEPHALUS

Under this heading is considered hydrocephalus associated with raised intracranial pressure from causes other than expanding lesions such as tumour, and abscess. In most cases the cause is obstruction in some part of the cerebrospinal pathway the result of a congenital membrane suppurative or non suppurative ependymitis or meningitis. In other cases there is an obstruction of the venous pathways through which the cerebrospinal fluid is absorbed and in this group is included hydrocephalus following thrombosis of the lateral or sagittal sinuses.

**Infantile Hydrocephalus**—The effects of hydrocephalus vary with the condition of the cranial sutures. In infants there is progressive expansion of the head and little or no papilloedema. As the ventricles dilate the cerebral cortex becomes thin the child loses its recently acquired functions such as walking standing sitting etc and often has epileptic convulsions. In time the roof of each orbit becomes flattened the eyeballs are displaced downward the optic nerves are compressed by the expanding cerebral hemispheres and the child goes blind. In some cases the hydrocephalus is not intense or

is spontaneously arrested and the child retains motor sensory functions but shows some degree of dementia.

In infants it is as a rule impossible to distinguish by clinical methods alone the cases of hydrocephalus due to congenital or acquired adhesions or to venous thrombosis from those due to tumour or pachymeningitis. The antecedent history and the presence of associated lesions such as spina bifida and von Recklinghausen neurofibromatosis may give clues as to the cause of the condition but in most cases such investigations are inconclusive. The radiological signs thinning of the cranial bones and



FIG. 154. Calcified ependymoma in a child aged 1½ years who showed considerable diffuse enlargement of her head. Case No. 3534.

separation of the sutures are present in all cases and it is only in the rare instances of infantile hydrocephalus due to calcified tumour (Fig. 154) that they are at all distinctive. The cause of the lesion and the site of obstruction are determined by ventriculography and by the injection of dyes. Methylene blue is injected through the anterior fontanelle into the lateral ventricle and after a few hours it is not recovered from the opposite lateral ventricle the

obstruction is at the foramina of Monro if it cannot be recovered from the spinal canal the obstruction is probably at or proximal to, the foramina of Magendie and Luschka, on the other hand, if the lumbar cerebrospinal fluid is tinted blue, the obstruction must be distal to the foramen of Magendie, either in the subarachnoid spaces, especially at the tentorial opening, or in the cerebral veins or sinuses

These methods of investigation throw considerable light on the site of obstruction, and occasionally also on the cause but it must be admitted that they are often inconclusive Furthermore, operative treatment even a rational short-circuiting operation designed to provide an alternative pathway for escape of the cerebrospinal fluid is rarely successful

**Adult Hydrocephalus**—In adults the same causes may prevail to produce hydrocephalus as in infants, even congenital membranes may be found in adults at the foramen of Magendie and elsewhere, associated with quite a short history of illness The symptoms however are vastly different from those found in infants the head does not expand, or only to a small degree and there are often severe headache and papilledema The symptoms, in fact are identical with the hydrocephalic symptoms occurring in cases of intracranial tumour The radiological appearances have already been described (Chapter I, page 21)

In a few cases a previous history of meningitis meningococcal or otherwise, mastoiditis, and lateral sinus thrombosis etc, may indicate the nature of the hydrocephalus, but in many cases it is impossible, even after full radiological studies with air, to be certain of the cause, and the solution of the problem must await disclosure at operation In adult hydrocephalus the results of decompression and of short circuiting operations are, as a rule, good

## BIBLIOGRAPHY

### General Reviews on Intracranial Tumours

- BAILEY, P. *Intracranial Tumours* 1933 London Bailliere, Tindall & Cox  
 CLAIRNS H. 'A Study of Intracranial Surgery, Medical Research Council Special Reports No 12, 1929 London H M Stationery Office, 'The Ultimate Results of Operations for Intracranial Tumours' *Yale Journal of Biology and Medicine*, 1936, VIII 421  
 CUSHING H. *Intracranial Tumours* 1932 Springfield Ill, C C Thomas  
 DANDY, W E. *Practice of Surgery* (Dean Lewis), 1932 XII Hagerstown Md, W F Prior  
 DAVIS, L. 'Intracranial Tumours' *Ann of Roentgenol*, 1933 XIV, New York Hoeber  
 LIMA ALMEIDA 'Contribuição para o Estudo da Circulação dos Tumores Intracranianos' 1938 Lisboa, der Silva  
 MONIZ, E. *Diagnostic des Tumeurs Cerebrales et Épreuve de l'Encephalographie Arterielle*, 1931, Paris Masson & Cie  
 NACHS, E. 'Diagnosis and Treatment of Brain Tumours' 1931, St Louis Mosby, and London, Humpston

## Pituitary Tumours

- CUSHING H Pituitary Body Hypothalamus and Parasympathetic Nervous System  
1932 Springfield Ill C C Thomas
- DAVIDOFF L M Studies in Acromegaly, *Endocrinology* 1926 X 461
- DOTT N M and BAILLY P A Consideration of the Hypophysial Adenomata *Brit Journ Surg* 1925 XIII 314
- HENDERSON W R The Pituitary Adenomata *Brit Journ Surg* 1939 XXVI 811

## Sellar and Suprasellar Epidermoid Tumours

- CRITCHLEY McD and IRONSIDES R N, The Pituitary Adamantinomata *Brain* 1926 XLIX 437
- McKENZIE K G and SOSMAN M C The Roentgenological Diagnosis of Cranio-pharyngeal Pouch Tumours *Amer Journ Roentgenol* 1924 XI 171

## Meningioma

- BAILEY P and BUCY P C The Origin and Nature of Meningeal Tumours *Amer Journ of Cancer* 1931 XV 15
- CAIRNS H The Radiological Appearances of Meningiomas *Proc Roy Soc Med* 1936 XXI 1168
- CUSHING H Meningiomas 1938 Springfield Ill C C Thomas
- LEHMAN F Cranial Osteomas and Hyperostoses produced by Meningeal Fibroblastomas *Arch Surg* 1934 XXXVIII 357
- HENDERSON W R The Anterior Basal Meningiomas *Brit Journ Surg* 1938 XXVI 124
- HORTEGA P DEL RIO Anatomia microscopica de los Tumores del Sistema Nervioso Central y Periferico 1933 Madrid
- ROWBOTHAM G F The Hyperostoses in Relation with the Meningiomas *Brit Journ Surg* 1939 XXVI 593
- SCHWARTZ C W The Meningiomas *Amer Journ Roentgenol* 1938 XXXIX 698
- SOSMAN M C and PUTNAM T J Roentgenological aspects of Brain Tumours *Amer Journ Roentgenol* 1925 XIII 1
- DE VET A C Over de Diagnostiek van het Meningioma Cerebri 1936 Amsterdam Sel eltima & Holkema

## Glioma

- BAILEY P Intracranial Tumours 1933 London Bailliere Tindall & Cox
- BAILLY P and CUSHING H Tumors of the Glioma Group 1926 Philadelphia Lippincott
- HORTEGA P DEL RIO Estructura y Sistematizacion de los Gliomas y Piraghiomas *Arch Espanoles de Oncologia* 1932 II 411
- ROUSS C and OBERLING C *Annales du Cancer* 1931 fasc IX and X Paris Felix Alcan

## Acoustic Neurinoma

- CUSHING H Tumors of the Nervous Acoustic 1917 Philadelphia W B Saunders
- MAYER E G Otologische Röntgenagnostik 1930 Berlin Springer

## Colloid Cyst of the Third Ventricle

- DANDA W E Benign Tumors in the Third Ventricle 1933 London Bailliere Tindall & Cox

## Cholesteatoma and Allied Tumours

- HAILEY, P. Further Observations on Pearl Tumours. *Arch of Surg* 1924, VIII 524
- CRITCHLEY, McD and FERCLSON F R. The Cerebro-spinal Epidermoids (Cholesteatomata). *Brain* 1928 LI 334
- CUSHING H. A Large Epidermal Cholesteatoma of the Parieto-temporal Region. *Surg Gynec and Obst*, 1922 XXXIV 557
- HORRAX C. A Consideration of the Dermal Versus the Epidermoid Cholesteatomas having their Attachment in the Cerebral Envelopes. *Arch Neurol and Psychiat* 1922 VIII, 275
- JEFFERSON G and SMALLER A A. Progressive Facial Palsy produced by Intratemporal Epidermoids. *Journ Laryngol and Otol* 1938 LIII 417
- PATTISON A R D. Supracalcaneal Epidermoid Cholesteatomata. *Lancet* 1937 II 1303

## Chordoma

- BAILEY P and BACDASAR D. Intracranial Chordoblastoma. *Irish Journ Path* 1929 V 439

## Papilloma

- DAVIS J F and CUSHING H. Papilloma of the Choroid Plexus. *Arch Neurol and Psychiat* 192 XIII 681
- VAN WAGENEN W P. Papillomas of the Choroid Plexus. *Arch Surg* 1930 XX 193

## Tuberculoma

- MARTIN J P. Calcified Intracranial Tuberculomata. *Brit Journ Radiol* 1937 X 5
- VAN WAGENEN W P. Tuberculoma of the Brain. *Arch Neurol and Psychiat* 1927 XXVII 57

## Cysticercosis

- MACARTHUR W P. Cysticercosis as seen in the British Army, with Special Reference to the Production of Epilepsy. *Trans Roy Soc Tropical Med and Hygiene* 1934 XXXVII 313

## Hydatid Disease

- DREW H R. Hydatid Disease of the Brain. *Surg Gynec and Obstet* 1934 LIX 321
- KNEPPOHN J F M. Hydatid Disease. *Med Journ of Australia* 1937 I 201

## Intracranial Aneurysm

- ALBRECHT F. The Syndrome Produced by Aneurysm at or near the Junction of the Internal Carotid Artery and the Circle of Willis. *Bull Johns Hopkins Hosp* 1929 XLIV 215
- BRADWELL F. Neurology. *Trans Ophthalmol Soc (U K)* 1934 LIV 205
- CARRON H. Tubercular Ocular Palsies from the Neuro-ophthalmic Point of View. *Proc Oxford Ophthalmol Soc* 1938
- DYER N M. Intracranial Aneurysms. Cerebral Arteriography. *Edin Med Journ*, 1933 XL 219
- FRANKSIDES F C. Intracranial Aneurysms. *Brain* 1916 XXXIX 224
- HERMANN K, ORRATORIO S and DYER N M. Intracranial Aneurysms and Allied Clinical Syndromes. Cerebral Arteriography in their Management. *Lisboa Med* 1937 XIV 782

- JEFFERSON G On the Saccular Aneurysms of the Internal Carotid Artery in the Cavernous Sinus *Brit Journ Surg* 1938 XXVI 267 Compression of the Chiasma Optic Nerves and Optic Tracts by Intracranial Aneurysms *Brain* 1937 LX 444
- NATTHASS F J Clinical Manifestations of Intracranial Aneurysms *Lancet* 1933 II 915
- RIDDOCK G and GOULDEN C B On the Relationship between Subarachnoid and Intraocular Hemorrhage *Brit Journ Ophthalm* 1925 IX 209
- SOSMAN, M C and VOLT L C Aneurysms of Internal Carotid and Circle of Willis *Amer Journ Roentgenol* 1926 XV 122
- SYMONDS C P Contributions to the Clinical Study of Intracranial Aneurysms *Gujst Hosp Reports* 1923 LXXIII 139
- TURNBULL H M Alterations in Arterial Structure and their Relation to Syphilis *Quart Journ Med* 1915 VIII 201

### Angiomatic Malformations

- BERGSTRAND H OLIVCRONA H and LOENNIS W Gefassmissbildungen und Gefassgeschwulste des Gehirns 1936 Leipzig Thieme
- CUSHING H and Bailey P Tumors arising from the Blood vessels of the Brain 1928 Springfield Ill C C Thomas
- DANDY W E Arteriovenous Aneurysm of the Brain *Arch Surg* 1923 XXII 190 Venous Abnormalities and Angiomas of the Brain *Arch Surg* 1928 XXVII 715

### Subdural Hæmatoma

- MARTIN J P Chronic Subdural Hæmatoma *Proc Roy Soc Med* 1931 XXIV 19

### Brain Abscess

- CAIRNS H Brain Abscess from the Point of View of the Neurological Surgeon *Proc Roy Soc Med* 1930 XXIII 1049
- CAIRNS H and DONALD C Diagnosis and Treatment of Abscess of the Brain *Proc Roy Soc Med* 1934 XXVII 1643
- DANDY W E Pneumocephalus of Bacterial Origin *Arch Surg* 1927 XI 913 Practice of Surgery, 1932 XII 370 Hagerstown Md W F Prior
- SYMONDS C P Some Points in the Diagnosis and Localisation of Cerebral Abscess *Proc Roy Soc Med* 1926-27 XX 1139
- SYMONDS C P and CAIRNS H Brain Abscess *Brit Encyclop of Med Practice* 1936 II 597
- VINCENT C Sur une methode de traitement des abcès subaigus des hémisphères cérébraux large décompression puis ablation en masse sans drainage *Gazette med de France* 1936 XLIII 93

### Hydrocephalus

- CAIRNS H Störung der Sekretion und Resorption der Cerebrospinalflüssigkeit und ihre Behandlung *Deutsche Zeitschr f Nervenheilkunde* 1935 CXXV III 180
- SYMONDS C P Hydrocephalus *Brit Encyclop of Med Practice* 1936 VI 566

## CHAPTER V

### ANOMALIES AND DISEASES OF THE SKULL

#### MALFORMATIONS, CONGENITAL AND INFANTILE <sup>1</sup>

Cyclops is a condition of fusion of the cerebral hemispheres and the eyes. There is one centrally placed orbit containing one or two eyeballs, and the nose is absent. The condition is not compatible with life. The term *arrhinencephaly* (absence of the rhinencephalon) includes lesser degrees of the same fusion malformation. Some of these are compatible with life to an advanced age. In the severe cases the two orbits are in contact with one another and there is no nose. Among the milder degrees of arrhinencephaly is *trigonocephaly* in which there is premature union of the two lateral masses of the frontal bone, the frontal region is narrowed and forms a keel-like projection. The cerebral hemispheres form a single mass, the lateral ventricles are fused at least in their anterior parts, and the corpus callosum is absent. Hare lip and cleft palate often occur and distant malformations are common including spina bifida and polydactyly. In patients who survive absence of sense of smell is invariable and epilepsy, psychotic symptoms and eunuchoidism are common. In minor degrees of arrhinencephaly air injection may be useful in showing absence of the septum lucidum and of the corpus callosum.



FIG. 15\* A large irregularly oval defect in the cribriform plate associated with a large meningocele extending to the root of the nose.

hernia may consist of the brain coverings only (*meningocoele*) (Fig 156) or of the brain coverings and a thin layer of brain substance (*cephalocoele*). The term *meningocoele* is often applied to both variations though actually the majority are *cephalocoeles* and if air is injected into the sac it is found



Fig 156 Congenital defect in the region of the sagittal suture associated with a meningocele. Male age 1  $\frac{1}{2}$  years. Case No 3067

by X rays to connect directly with the ventricular system. *Spina bifida* is a common accompaniment. **Microcephaly**, in which the sutures are normal must be distinguished from *craniostenosis* in which there is premature closing of the sutures. In *microcephaly* underdevelopment of the vault of the skull is secondary to underdevelopment of the brain. It may be congenital (*microcephaly vera*) or due to some disease which stops the growth of the brain in infancy or childhood. In the latter conditions the head is small but shows no other changes. A small head alone does not constitute *microcephaly*; the size of the cranium must be considered in relation to the size of the face and the rest of the body. In true *microcephaly* the forehead slopes backwards to a flattened vault; usually the occiput is nearly

normal in size and shape. The bones of the face are average in size; the nose is long. It has been affirmed that owing to the poor growth of the brain the bones of the skull may even overlap or an excess of fluid may be found between the brain and the skull. The bones of the vault are often thickened. The condition may be familial. **Craniostenosis** comprises a group of conditions in which the most important feature is closure of the cranial sutures in infancy or even before birth. The head may assume various shapes from which the condition is subdivided into a number of types. Closure of the sutures seems to be the primary lesion and symptoms arise from insufficient room for the developing brain. There is severe rise of intracranial pressure and a characteristic radiological feature in these cases is that there are very deep and numerous convolutional markings. *Oxycephaly* (Syn *Steeple Head* *Turnschadel*) is the most common variety of *craniostenosis*. It is sometimes hereditary and familial. The head is small and abnormally high. The coronal suture is usually closed

but the parieto-occipital suture is not often involved. Digital impressions are very pronounced. The floor of the middle and anterior fossæ is frequently depressed, and patients with this condition show considerable proptosis. The sella turcica may be normal, but is often deepened and widened. The condition is compatible with normal intelligence, but not infrequently optic atrophy supervenes and may even advance to complete blindness. In some cases this blindness may be due to kinking of the optic nerves at the optic foramina, which may themselves be narrowed, in other cases it may be due to rise of intracranial pressure. *Scaphocephaly* (Syn. *boat-shaped head*) is usually secondary to closure of the sagittal suture, so that growth takes place in the sagittal plane, but not in the transverse diameter. Thus a long, boat-shaped head is produced, the closed sagittal suture representing the keel. *Plagiocephaly* (Syn. *oblique or flattened head*) is caused by closure of the sutures on one side of the skull retarding development on that side and producing an asymmetrical or slanting skull.

**Basilar Invagination.**—In this condition there is upward displacement of the foramen magnum, and the consequent deformity of the base of the skull may bring about symptoms of ponto bulbar and cerebellar disturbance, together with compression of cranial and upper spinal nerves, and also hydrocephalus. In the majority of cases the cause is not evident, though any condition producing lack of rigidity of the bones of the posterior fossa, such as osteomalacia, fragilitas osseum, and other deficiency diseases, may result in collapse of the head upon the atlas vertebra and diminution in the size of the posterior fossa.

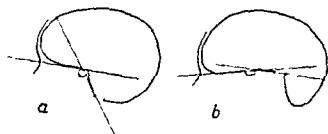


FIG. 157. The basal angle. (a) normal (b) in basilar invagination.

The basal angle<sup>1</sup> normally 115-140 degrees, approximates to 180 degrees (Fig. 157). The foramen magnum is displaced upwards into the base of the brain to such an extent that the odontoid process of the axis may reach the region of the dorsum sellæ and press on the medulla (Fig. 158). This deformity and the effect of gravity cause the atlas to ride forward and rest on the base of the skull anterior to its normal position. Changes are found in the upper cervical vertebrae, the atlas may be fused with the base of the skull or with the axis. The floor of the posterior fossa is thin and long.

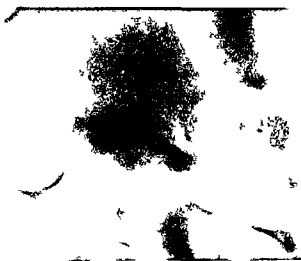
**Osteogenesis Imperfecta** (Syn. *Fragilitas ossium*)—Inability to form osteo-

<sup>1</sup> The basal angle is the angle formed at the point of intersection of a line from the anterior point of the foramen magnum to the centre of the sella turcica, and a line from that point to the posterior point of the foramen magnum.





(a)



(b)

FIG. 158.—Larval mandible. (a) Anteroposterior projection. (b) Lateral plan. The upper cervical teeth and the basipharyngeal region are displaced upwards to such an extent that the anterior part of the foramen magnum lies above the level of the crests of the petiole bones. There is gross flattening of the basipharynx. (Through the kindness of Dr. A. de Vries and Dr. B. G. Zeddes des Plantes.)

blasts results in weakness and bending of bones. In any condition where ossification is incomplete and the bones are soft such as *fragilitas ossium*, osteomalacia, rickets and hyperparathyroidism gravity may cause the skull to sag over the cervical vertebrae. It is obvious that the unsupported occipital region will be more affected than the anterior part of the base of the skull which is supported by the facial bones arranged in many different planes and layers. The occiput tends to sag over the nape of the neck and the parietal and temporal regions tend to sag over the ears. The appearance has given rise to the name *tam o shanter skull*. Large areas of the bones of the vault remain unossified and there is delay in closure of the sutures. Large numbers of Wormian bones appear.

**Cerebral Diplegia (Little's Disease).**—In the spastic paralysis of infants usually due to injury at birth various abnormalities of the skull may be found. Asymmetry of the skull may arise from arrest of development of one cerebral hemisphere (Fig. 150) and corresponding arrest of growth of the overlying cranial bones. In other diplegic children diffuse hyperostosis of the frontal and parietal bones has been observed.

In some varieties of cerebral atrophy of infants great thickening of the skull bones especially of the base of the skull has been found.

## ENDOCRINE AND CONSTITUTIONAL DISEASES

Abnormalities of the skull due to endocrine and constitutional diseases comprise a group of clinical states that are as yet for the most part poorly defined. They include conditions in which the changes in the skull are part of a general osseous disease and other conditions in which the skull is the only part of the osseous system affected. In most of them the pathological changes are not limited to the bones with the growth of knowledge more and more of these diseases are shown to be due to primary changes in the soft tissues. The classic example of this is the discovery that acromegaly and acromegalic gigantism are due to tumour of the pituitary gland. In more recent years important additions to our knowledge of bone pathology have come from discoveries of the relationship of vitamin D deficiency to rickets and osteomalacia and of the connection between generalised osteitis fibrosa and tumour of the parathyroid gland. There are however many abnormal states of the skull in which the cause is still unknown and these increase in number with the increasing popularity of radiology of the skull. Recently *E. Mellanby* has shown that young dogs fed on diets deficient in vitamin A and rich in cereals develop overgrowth of the base of the skull including the bony labyrinth with resulting deafness.

Most of the conditions considered in this section are diseases in which there is diffuse bony thickening of the skull and in this connection it is important to recognise that the radiographic appearances of the skull are usually not specific. This is not surprising when we recall that even the bone changes as viewed under

the microscope, may not be specific, thus, the bony changes of generalised osteitis fibrosa, with its associated parathyroid tumour and characteristic alterations in blood chemistry, are identical with those of a condition called localised osteitis fibrosa, in which there is no parathyroid tumour and no known disturbance of body chemistry. Even when the histological changes are dissimilar, the X ray appearances of the skull may be alike. In a case of thickened skull it may be impossible to differentiate between osteomalacia, generalised osteitis fibrosa, osteitis deformans, secondary carcinomatosis, and the woolly type of renal infantilism, without recourse to radiographic examination of other bones and full clinical and chemical studies. The differential diagnosis of such conditions is considered fully in the chapters on constitutional diseases and diseases of unknown origin (Chapters XXXVIII, XL, XLIII and XLIV).

**Dwarfism and Infantilism**—In the racial dwarfs and small people, such as the Pygmy tribe of the Belgian Congo, and the Gurkhas, the skull is generally small, but normal in all other respects. In the *pituitary dwarf* (Loiran type) there is deficiency of the anterior lobe of the pituitary, this may be associated with epidermoid tumours of Rathke's pouch. The skull is small and of childish shape. The teeth are often unerupted. The sutures tend to remain open. The sella turcica may be within normal limits in size, but, if an epidermoid tumour is present, it may be enlarged. In *cretinism* the base of the skull is short, while the whole skull and face are wide. The skull bones are thick, but ossification and eruption of teeth are commonly delayed. The pituitary fossa is usually unchanged, but may be enlarged. Wormian bones are common.

In *mongolism* delayed ossification is seen, and Schuller reports circular defects in the line of the sutures. In *achondroplasia* the os triasulare, consisting of the pre- and post-sphenoid and basi-sphenoid nuclei, undergoes premature fusion, and in consequence the base of the skull is short for the size of the vault. The root of the nose is depressed and the posterior fossa shallow. The sella turcica is normal in size, but the direction of the clivus is almost vertical. The lower jaw is normal in size. The eruption of the teeth is not delayed.

**Gigantism**—Excessive growth may be racial and give no pathological manifestations. The eunuchoid giants show non-union of the epiphyses, and, at times, slight enlargement of the pituitary fossa. The cranium is small compared with the rest of the body and is sometimes thickened. There is *delayed eruption of the teeth*. The face is usually long.

**Acromegaly and Acromegalic Gigantism** have been described in Chapter IV.

**Generalised Osteitis Fibrosa (Syn. Hyperparathyroidism)**—The skull as a whole is thickened and shows a fine mottled appearance, with islands of normal bone surrounded by areas of relative translucence. The contours of the inner and outer tables are less sharp than normal. Radiologically the condition may so closely resemble osteitis deformans that differential diagnosis can only be made by estimation of the blood calcium. Cyst-like areas may be seen in the vault.

**Hyperostosis Cranii**—The term hyperostosis cranii has been applied to a variety of bony lesions including localized expansions of the cranium due to meningioma of the vault or base neoplasms of the skull osteitis deformans osteitis fibrosa and to chronic periostitis. There is also a special variety of hyperostosis of the inner table of the frontal bone (*hyperostosis frontalis interna Morgagni's syndrome*) which occurs usually though not invariably in stout women at the menopause especially in those who show excessive hair on the face (Fig. 159). There is an irregular proliferation of the inner table of the frontal bone which occasionally extends to the anterior parts of the parietal bones but not to the orbital roof. The condition is not associated with



FIG. 159.—Hyperostosis frontalis interna associated with an internal hyperostosis of a meningioma of the left parietal bone (marked with arrow). In addition the patient had a meningioma of the right optic nerve sheath (arrow of which can be seen in the right orbit). Stout woman aged Case No. 166.

clinical symptoms and does not indicate any disease within the cranial cavity. It is probably due to disturbance of the endocrine system and *Folle Henschen* has found increase of the granular cells and diminution of the chromophilic cells of the anterior lobe of the pituitary gland. The dura becomes firmly adherent to the areas of hyperostosis and in osteoplastic exploration of the cranium of the frontal region great difficulty will be encountered in separating the dura from the overlying bone.

A similar variety of frontal hyperostosis is seen in old people associated with thickening of the whole of the cranial vault and atrophy of the cerebral convolutions. Other types of hyperostosis of the calvarium have been described by *Sternrood Moore* including hyperostosis calvaria diffusa the e

are more common in women than in men. During pregnancy small hyperostoses are sometimes formed on the inner table of the skull.

A peculiar type of hyperostosis localised to one half of the skull and face has been described by *Brissand Leri Brouwer* and others under the name of *Iemicrocranium*. Numerous exostoses project from the inner and outer tables both of the vault and base on one side. On the same side there may be numerous meningiomas attached to the dura and the underlying brain may show hypoplasia and degenerative lesions.

**Rickets and Osteomalacia**—In gross cases of vitamin D deficiency the skull together with all the other bones of the body shows pronounced changes as a result of the delay of calcification of the osteoid tissue. In rickets normal new bone is not laid down at the growing edges of the bones and the sutures and fontanelles of the vault remain widely open. The bones are thin (craniotabes) and deficient in calcium. In infants the bones may be so soft that the head becomes flattened where it rests on the pillow. The upward displacement of the cervical vertebrae and the posterior part of the base of the skull known as basilar invagination (p. 167) may be the result of rickets in early life. Deposits of osteoid tissue may occur on the outer surfaces of the parietal and frontal bones. The bones of the face do not develop normally, eruption of teeth is delayed and the palate is narrow and highly arched.

Osteomalacia affects the adult only and therefore the deformities due to interference of growth are not seen. The outstanding feature is lack of calcium with deformity due to gravity or pressure in extreme cases. At times the cranial vault may show scattered areas of uneven translucency.

**Idiopathic Steatorrhœa (Syn. Cœliac Disease)**—In this condition fully described in Chapter XXXVIII the cranial bones are deficient in calcium and there is delay in closure of the suture lines between them. Irregular areas of osteoporosis similar to those seen in osteomalacia may appear. We have no record of marked deformity in this condition.

**Renal Infantilism**. In the *rachitic* type of this disease no changes apart from those consequent on the delayed closure of the sutures are observed. In the *woolly* type (Chapter XL) the bones of the cranium may be thickened and widened and may contain circular areas of translucency which vary in size. The bones of the base of the skull may be affected.

**Xanthomatosis (Syn. Schuller-Christian Disease)**—In this condition the bone is replaced by a lipid substance, cholesterol. Small foci arise in the skull and spread irregularly, gradually becoming confluent or one area enlarges until large tracts of the skull have become involved. The edge of the defect is clean cut and shows no sclerosis (Fig. 456). In radiograms no debris is visible in the translucent area. Both tables of the skull are diseased. The appearance is aptly described as being like a map. The base of the skull is often involved especially the anterior fossa and anterior part of the middle fossa. If the orbital roofs are affected there is proptosis. Deposits of lipid

are not limited to the bone but may be present in the soft tissues especially around the stalk of the hypophysis with the production of diabetes insipidus. We have seen a case of xanthomatosis affecting the iliac bone proved by histological examination in which polyuria was the presenting symptom and in which no bony changes were visible in the skull until several years later. Sosman has demonstrated the prompt therapeutic action of X rays on these lesions. He was able to prove that this action was a direct one by covering part of an invaded area with a lead shield before treatment with the result that only the exposed part healed.

**Erythroblastic Anæmia (Cooley)**—This disease chiefly affecting children

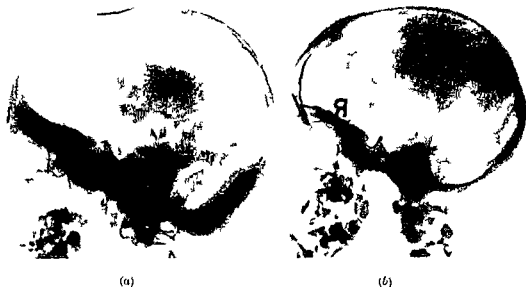


FIG. 160—(a) Gross thickening of the base of the skull in a man who had a spontaneous fracture of the femur. Osteitis fibrosa cystica was confirmed by biopsy. (b) The skull from a case of marble bones (osteopetrosis) showing great increase of density and considerable thickening of the base of the skull. (Other bones from the same patient are illustrated in Fig. 477 and 48.)

of Mediterranean parentage is characterised by anaemia, hepatosplenomegaly and a general lack of calcium throughout the skeleton. In the skull there is widening of the diploe and in the later stages the vault is thickened and the outer table is replaced by radiating spicules of bone.

**Focal Osteitis Fibrosa**—In this condition which affects also long bones (Chapters XLIV and XLVII) cysts may form in the bones of the face and jaws and the base of the skull may be affected but the vault is rarely if ever involved. In the base of the skull especially in the region of the sella turcica there may be very marked increase of density and thickness (Fig. 160a). Care must be taken not to confuse this disease in the cranial base with the hyperostosis seen with some meningiomas. In osteitis fibrosa the bone is uniformly

thick and dense over a fairly wide area and lacks the transverse striation so often seen in hyperostosis of meningioma. The lesions may be multiple and cannot be distinguished from those of generalised osteitis fibrosa, except by absence of changes in the calcium content of the blood.

**Osteitis Deformans**—Osteitis deformans of the skull affects chiefly the outer table. Islands of dense bone appear on the outer surface of the skull, the bones of the vault thicken, and the differentiation between the two tables becomes lost. In extreme cases the outline of the inner table disappears also so that the skull appears to consist of uneven dense patches with more translucent pits between them (Fig 483, Chapter XLIV). Cyst like areas may be seen. A similar appearance has been described in renal infantilism. The condition may occasionally be limited to part of the cranial vault (Fig 34). The bony lesion may become sarcomatous. *Osteoporosis circumscripta*, in which large areas of clean cut osteoporosis form, is an early manifestation of osteitis deformans. In appearance it is somewhat like xanthomatosis, but on careful examination coarse trabeculation is seen in the translucent area. *Osteoporosis circumscripta* may be mistaken for a metastatic deposit, or for invasion of the skull by direct spread of a neoplasm of the scalp.

**Marble Bones** (Albers Schonberg's Disease) is a constitutional disturbance of bone development which begins in foetal life. All the bones of the skeleton become thick and extremely dense to X rays. Spontaneous fractures occur in the long bones. The condition may be familial. In the skull the base is affected more than the vault. All the accessory sinuses are obliterated and the foramina are narrowed. Optic atrophy and blindness may follow involvement of the optic nerves and facial paralysis and nerve deafness may also occur. Radiologically the appearances of the skull may resemble those seen in certain cases of focal osteitis fibrosa (Fig 160b). The appearances of the vertebrae of marble bones are, however, diagnostic (Chapter XLIII).

### INFECTIONS OF THE SKULL

**Pyogenic Osteitis**—Pyogenic organisms may reach the skull by an open wound following trauma, by the blood stream, or by spread from the scalp accessory sinuses and mastoid cells. In acute infection, the earliest date at which there is any possibility of detecting a change in the radiograms is fourteen days after the beginning. If the infection is not virulent there may not be detectable signs even at this date. The first radiological manifestation is an area or a group of small areas of extra translucence, as these increase in size they are seen to be uneven in density and irregular in outline. They spread and become confluent (Fig 161). The infection follows the line of least resistance and destroys the smaller trabeculae of the bone more readily than the coarser, in contradistinction to a neoplasm which destroys equally in all directions.

In chronic infection, in addition to destruction there is marked thickening

and sclerosis, the appearance may be one of extra density containing areas of translucence. Sequestra may form and be seen as fragments of very dense bone lying in a zone of extra translucence. If a tangential view is taken, small fragments of detached bone may be seen. The outline is almost always irregular and uneven. The petrous, squamous temporal, and parietal bones are most commonly attacked. Osteomyelitis will often be limited to one bone and the sutures appear to constitute a line of resistance, especially in the young.

**Syphilitic Osteitis**—As a result of modern treatment tertiary syphilis is rapidly becoming a rare malady and it is uncommon to see bone manifestations



FIG. 161.—Pyogenic osteomyelitis of the vault of the skull two months and (small inset) six months after the on-set of symptoms. Case No. 1539.

In the congenital form the saddle back nose results from erosion and malformation of the nasal bones and bones in the base of the skull. In the late congenital and in acquired syphilis the vault of the skull may slowly become thickened and sclerosed over a wide area. In other cases single or multiple areas of necrosis may occur and these may be surrounded by sclerotic bone.

**Tuberculous Osteitis**—Tuberculous disease of the skull is rare, and we have never seen a verified case. Schuller describes it in his monograph and Strauss has recently reviewed the literature and described three cases.

**Radium, or X-radiation Necrosis** is occasionally seen after excessive radiation treatment of malignant tumours of the scalp. The necrotic areas consist of dense bone of serpiginous outline, and are surrounded by an irregular zone of diminished density, representing the granulation tissue that is attempting to absorb the dense bone.



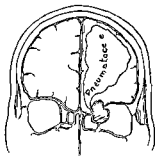


FIG 16 Orb to ethmoidal osteoma with secondary arachnoid cyst of the frontal lobe (After C. S. L. G.)

## TUMOURS BENIGN AND MALIGNANT

**Osteoma**—These bony tumours may arise from the outer or inner table of the skull but if large may involve both tables. On the outer table they are generally shallow convex masses of bone which may be composed entirely of ivory bone or may contain cancellous tissue which is continuous with the diploic layer. Osteomas that project into the cranium arise for the most part in the ethmoidal and frontal sinuses and also occasionally on the roof of the orbit or surface of the petrous bone. They are usually lobulated and even pedunculated and consist of dense ivory bone. Their radio-

graphic appearance is thus characteristic (Fig 162). As they grow intracranial osteomas tend to perforate the dura and even to embed themselves in the brain. It is important to note that small osteomas in the ethmoidal region may thus give rise to serious intracranial symptoms: these include proptosis of one eyeball, epileptic attacks, cerebrospinal rhinorrhoea and purulent leptomeningitis, also intracerebral arachnoid cyst, a collection of air in the frontal lobe which is visible in radiograms (Fig 162).

**Hæmangioma**—Cavernous hæmangiomas of the skull are not uncommon. They are benign tumours of slow growth and are for the most part small: they expand the bone giving rise to a hard and at times painful lump on the head but seldom to any intracranial symptoms. On clinical grounds it is not always easy to differentiate between this condition and the hyperostoses of meningiomas. Radiologically however the distinction can sometimes be made for the hæmangioma appears as a sharply defined area of diminished density traversed by fine trabeculations (Fig 163). Tangential views may show

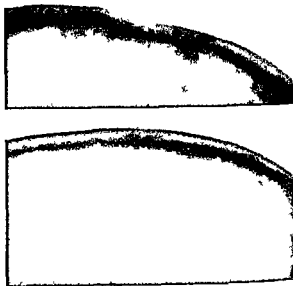


FIG 163 Hæmangioma of the right parietal bone  
Ca 6 No 2 60

fine radiating spicules of bone in the tumour. These tumours are not associated with cerebral haemangiomas and do not recur after removal.

**Chordoma** a tumour arising from the remains of the cranial end of the notochord grows posterior to the clivus and extends intracranially and also at times invades the basi sphenoid or basi occiput (see Chapter IV).

**Other benign tumours of the skull** include *cholesteatoma* a *myeloma* of the plasma cell type single or multiple (Fig. 164) and *simple cyst*.

**Sarcoma**—Sarcoma of the skull usually expands outwards more than inwards. It is a tumour of rapid growth producing an osteogenic reaction of only moderate intensity. New bone is laid down in radiating spicules at right angles to the bone surface (Fig. 35) but often can only be demonstrated in transverse views by means of a very soft radiation. Indeed a diagnostic feature of importance is the fact that with a large lump on the head ossification in sarcoma is too incomplete to show in radiograms taken with ordinary penetration whereas in other tumours it is usually intense. There may be considerable patchy destruction of the skull in the area of attachment of the tumour. While most of these tumours are found in the vault of the skull some occur in the base or invade the base from the adjacent nasopharynx and air sinuses. Sarcoma of the skull is usually a primary disease but may supervene in areas of osteitis deformans or of focal osteitis fibrosa.

**Metastatic Tumours**—Metastatic tumours of the skull may show as an area of increased or diminished density and may be multiple or single. Solitary metastases may grow to large size and at times so slowly that it is difficult to distinguish them without histological examination from meningioma (Fig. 165). Metastatic carcinoma of the thyroid grows in this manner at times. Primary neuroblastoma of the adrenal medulla of children frequently metastasise to the



FIG. 164. Large area of erosion on left table of frontal bone. Large plasma-celled myeloma. Case No. 3359.

skull and may be associated with proptosis of the eyeball on the corresponding side. The Grawitz tumour of the kidney also not infrequently metastasises to

the skull and the other primary sources of skull metastases may be in any of the usual sites. Without biopsy multiple metastatic tumours may be difficult to distinguish from multiple myeloma.

**Carcinoma of Direct Spread** — Carcinoma may involve the base of the skull by direct spread from the nasopharynx or air sinuses. Carcinoma of the pharynx may invade and destroy the petrous bone and produce symptoms of a cerebello pontine angle tumour. The primary carcinoma may be so small that it is easily overlooked



FIG. 163.—Metastatic carcinoma proved by histological section at the highest point of the vertex. Primary tumour undetectable. The bone changes start as a slight enlargement. Definite lump present. Man, age 196. Case No. L.H. 1846. O.P.

and in consequence in some cases of the cerebello pontine angle syndrome the radiographic evidence of destruction of the petrous bone may be particularly important in diagnosis. Carcinoma may also affect the cranial vault as a result of spread of a rodent ulcer (basal celled carcinoma) of the face or scalp.

### FRACTURES OF THE SKULL

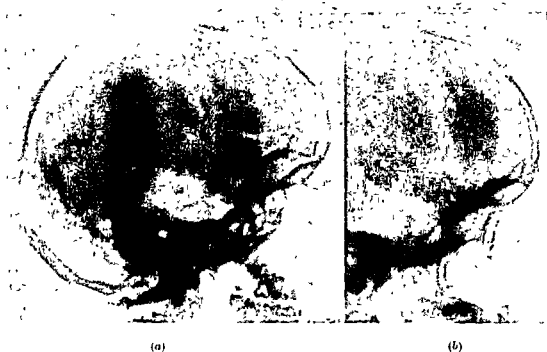
In head injuries it has long been taught that it is the damage to the cranial contents rather than injury to the skull that is of primary importance. For this reason and because of the difficulties of radiography in the acute stages of head injuries there has been a tendency to disregard the importance of radiography in head injuries except when medico legal considerations are at stake. No one would deny the truth of the teaching but detailed study of head injuries in recent years has shown that there are many ways in which radiography can contribute to diagnosis and treatment in the acute stage of head injuries. Among these the following may be specified.

(1) The depth and extent of comminuted and depressed skull fragments can be accurately assessed only by radiography. This is a help particularly

in closed head injuries (i.e. injuries in which the scalp is intact), in determining whether the dura is torn and operation thus necessary; and it is a guide to the surgeon when he comes to remove depressed bony fragments from the brain.

(2) Demonstration of the presence of a fracture indicates the site, or one of the sites, of maximum brain damage, a fact that often cannot be determined by clinical methods in an unconscious patient.

(3) A line of fracture crossing the channel of the middle meningeal artery arouses suspicion of an extradural middle meningeal hæmorrhage, a condi-



(a)

(b)

FIG. 10b. — Simple fissured fracture of the frontal bone, extending back on each side into the parietal bones. (a) Immediately after the fracture. (b) Twelve months later. Case No. 2228.

tion which is sometimes not recognised until it is too late to save the patient by operation.

(4) Fractures into the frontal and ethmoidal sinuses can be shown, and may call for operation to repair the dural defect and thus to prevent meningitis. In the same injuries air may be revealed inside the cranium, usually in the subdural space (Fig. 65).

It cannot be denied that many patients with head injury are so shocked or so restless when admitted to hospital that any elaborate radiographic examination is out of the question. But good organisation of the X-ray department, particularly the presence of a resident house officer in radiology, and the occasional resort to a good portable plant, overcome this difficulty to a large extent, and furnish knowledge of great value in the detailed study of head injuries.

Many fractures escape notice through the initial injury being too slight to warrant attention. *Stewart* found that in 300 cases examined after a history of head injury there was radiological evidence of fracture in 20 per cent. The true incidence of fracture in such a group is probably higher, because in the base of the skull necropsy shows many small fractures that cannot be adequately displayed by present radiographic technique.

The different types of fracture met with in the skull are (i) linear (ii) depressed and (iii) punctured.

**Linear Fracture** is the most common. It may be confined to the vertex or the base or may run from one to the other. The line of fracture may be in

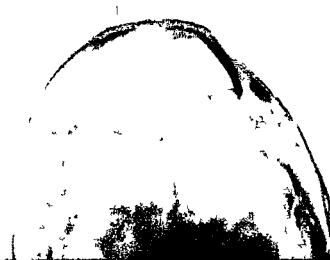


FIG. 167.—Tangential view showing the inward displacement of the fragments of bone in a patient with a star-shaped fracture of a vault.

any direction and the fissure will vary from a line like a hair to even 2 cm in width (Fig. 166). Gross fractures are obvious, but the short narrow linear fracture may at times be confused with suture lines, or with the channels of meningeal or diploic vessels. Sutures may resemble lines of fracture in the temporo-parietal region of young subjects in whom the serrations of the suture lines are poorly developed. The course of the temporo-parietal suture is, however, constant, and narrow fissures in that

region may be recognised by deviation from that course. It must be borne in mind that both in infants and adults fractures of the skull sometimes run along the lines of suture especially the coronal suture, and are in fact separated sutures. They can be best recognised by the separation of the edges seen in tangential projections of the suture.

Distinction between linear fractures and meningeal channels may be at times very difficult. The fracture usually has a more decisive and clean cut image, since it involves the whole width of the skull, whereas the meningeal channel is limited to the inner table. Lines of fracture run in any direction often change direction abruptly, and branch irregularly, whereas meningeal channels run in constant course, change direction gradually and branch dichotomously. Islands of bone surrounded by dark lines suggest a frac-

ture and in this connection it is important to bear in mind that though the meningeal artery and vein usually run in the same groove they may part for a short distance and then rejoin thus giving the impression of a splinter this appearance is most common in the proximal part of the meningeal channels that is to say near the pterion in which region fortunately the vascular channels are usually tortuous in outline and thus unlike a linear fracture Diploic vessels branch irregularly alter their course abruptly and may cross sutures lines just as fractures do but they can usually be distinguished from fractures by their uneven calibre which in some part of their course usually amounts to a beaded appearance

Even with all these points of differentiation it may sometimes be difficult to decide whether a small linear fracture is present The cases in which radiological controversies arise are usually more of medico-legal than of clinical interest and are best settled by repeating the X-ray examination with a series of specially designed stereoscopic and tangential projections

Depressed Fractures are always comminuted and may be circular or stellate If circular the whole plaque of bone is usually displaced inwards at one part of its circumference so that there is overriding of the edges in this part A few sharp flakes of bone are seen about the circumference of the

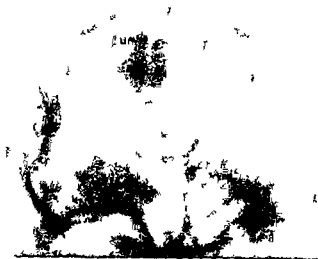


FIG 118 (a)—Depressed fracture of right parietal bone which was followed by attacks of epilepsy Case No 3115

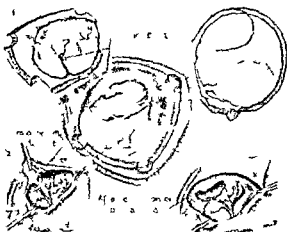


FIG 118 (b) Approaches to operation (1) The internal aspect of the bone flap showed a mass of depressed bone with irregular edges of the fracture and a corresponding indentation of the brain The inset (2) shows the internal aspect of the bone flap after it has been removed

Depressed Fractures are always comminuted and may be circular or stellate If circular the whole plaque of bone is usually displaced inwards at one part of its circumference so that there is overriding of the edges in this part A few sharp flakes of bone are seen about the circumference of the

main fragment (Figs 167 and 168) In stellate fracture the centre is usually depressed, there may be overlapping of the edges of some of the fragments

**Punctured or Penetrating Fractures** are the result of a blow from a small object such as a bullet The fracture may be complete or incomplete The usual finding with an incomplete fracture is detachment of a flake of the inner table and this may be difficult to detect

**Aerocœle** may be present if the fracture communicates with the atmosphere either directly or through the nasopharynx accessory sinuses or ear In this case there is bound to be escape of cerebrospinal fluid *Stewart* reports in one patient the escape of 38 oz. of fluid in twenty one hours after an injury

### Technique of Examination of Fracture of the Skull

Except in selected cases it is questionable if the routine radiographic examination of head injury by a portable ward apparatus is a reasonable procedure The detail of a ward radiogram is never of the best quality and in the early stages it is usually impossible to obtain pictures in any positions except the lateral and antero posterior After forty eight hours the risk of hæmorrhage is past and the majority of patients can be sent to the radiological department If a ward examination is unavoidable a Lysholm grid should not be used as it will lengthen the exposure and the grid lines may mask a fine linear fracture For routine examination all films should be stereoscopic and a fine focus tube and Bucky diaphragm are advised Antero posterior postero anterior and both lateral views should be taken The importance

of lateral views from both sides has already been pointed out If a depressed fracture is detected or suspected a tangential view will not only confirm the diagnosis but will give a fairly accurate estimate of the degree of depression Care must be exercised to get a true tangential view, otherwise the surgeon may obtain an erroneous impression Fractures of the base are very difficult to demonstrate and a high percentage escape detection The stereoscopic sub



FIG 169 —Old depressed fracture of the right parietal bone in a man aged 34 due to injury at the age of 4 years Case No 87

mento vertical view is of most value. As there is risk of considerable vascular congestion in the cranial cavity from the posture necessary for this picture, it should not be attempted until some time after the initial injury.

### Repair of Fractures of the Skull

Union of fractures of the skull is slow as compared with that of other bones, and there is little evidence of formation of callus. The extent of healing depends on the apposition of the fragments, the situation of the fracture and the age of the patient (Fig 166). Fractures of the base seem to heal more rapidly than those of the vault. The younger the patient, the better the healing. A simple fissured fracture in a child may no longer be visible in radiograms taken six months after injury. In adults most fissures are still faintly visible three years after the accident. Where there is not perfect apposition as, for example, in depressed fractures the edges of the bone become sclerosed and lose their irregularity. They are permanently visible in radiograms as edges of increased density, and may simulate radiologically a meningioma (Figs 28 and 169). Even when no bony union takes place, the space between the edges of the fracture is occupied by dense fibrous tissue and the skull is not excessively vulnerable on account of an old fracture. Where there is a gap in the skull, as a result of a comminuted fracture, bone tends to regenerate on the outer surface of the dura, especially in children. In radiograms of children who have previously undergone subtemporal or suboccipital (cerebellar) decompression, flakes of new bone are frequently seen in the coverings of the decompression (Fig 62).

### Birth Injuries

The majority of birth injuries appear as dents in the skull with no visible fracture, these dents fill out soon after birth. If a fracture is present in the dent it is usually of the "green stick" variety. Occasionally the dent or groove will persist, as in a case of injury to the frontal bone, which one of us examined repeatedly for over ten years.

### BIBLIOGRAPHY

- BROUWER, B. BIELSCHOWSKY M. and HAMMER I., 'Étude anatomo clinique d'un cas d'hémicraniose,' *Ann d'Anat Pathol*, 1936, XIII, 1.
- CANAVAN, M. M., Enostosis of the Calvarium *Arch Neurol and Psychiat* 1938, XXXIX, 41.
- CUSHING, H., Experiences with Orbito-ethmoidal Osteomata having Intracranial Complications *Surg Gynec & Obstet*, 1927, XLIV, 721.
- FERSTL, B., The Roentgen Appearance in Four Cases of Basilar Impression, *Acta Radiol*, 1934, XV, 652.
- ECHLIN, F., Cranial Osteomas and Hyperostoses produced by Meningeal Fibroblastomas *Arch Surg*, 1934 XXVIII, 357.
- GRIGG, D. M., Hypertelorism *Edin Med Journ* 1924 XXXI, 560.
- HENSCHEN, F., Morgagni's Syndrome, '1937, Jena G. Fisher.
- MELLANBY, E., The Experimental Production of Deafness in Young Animals by Diet, *Journ Physiol*, 1938, XCIV, 380.



- MOORE, S. "Metabolic Craniopathy," *Amer Journ Roentgenol*, 1936, XXXV, 30
- ROWBOTHAM, G. F. "Epidermoids arising in the Diploe of the Bones of the Skull," *Brit Journ Surg*, 1939, XXIV, 506
- SCHULLER, A., "Roentgen Diagnosis of Diseases of the Head," 1918, St. Louis, C. V. Mosby
- SEAR, H. R., "Some Notes on Craniostenosis," *Brit Journ Rad*, 1937, X, 445
- SKIPPER, E., "Oxycephaly," *Quart Journ Med*, 1934, XXVII, 579
- SOSMAN, M., "Xanthomatosis," *Amer Journ Roentgenol*, 1930, XXIII, 581
- STEWART, W. H., "Skull Fractures," *Ann of Roentgenography*, 1925, VI, New York, Hoeber
- STRAUSS, D. C., "Tuberculosis of the Flat Bones of the Vault of the Skull," *Surg Gynec and Obstet*, 1933, LVII, 384
- TAYLOR, JULIAN, "Invasion of the Skull by Dural Tumours," *Brit Journ Surg*, 1928, XVI, 6

## CHAPTER VI

### THE SPINAL CORD

It is only recently that substantial progress has been made in the diagnosis and localisation of spinal cord tumours by radiological examination. In 1924 *Pancoast* stated that tumours of the spinal cord could not be diagnosed by this method but as a result of the work of *Camp*, *Elsberg* and *Dyle* and others it is now claimed that 70 per cent. of all tumours of the spinal cord can be recognised radiologically.

In the majority of cases of spinal tumour the clinical picture is one of progressive weakness of one or more of the extremities associated with sensory and reflex disturbances and with pain which follows the distribution of spinal nerve roots. From the clinical examination combined with lumbar puncture and Queckenstedt's test it is possible in most cases to be reasonably certain that there is a tumour and the upper level of sensory loss usually, though not invariably indicates its approximate level. In many cases radiology adds so much to the precision of diagnosis that its use in suspected cases of spinal tumour has become indispensable. The methods comprise simple radiographic examination and radiographic examination after intrathecal injection of lipiodol. We will consider simple radiographic examination first.

#### TECHNIQUE OF SIMPLE RADIOGRAPHIC EXAMINATION

A Buel's diaphragm and fine focus tube are essential. The films should be well exposed as the laminæ and pedicles are not properly seen in under exposed or under penetrated pictures. The best view of the cervical region is obtained with the tube at 4 or 6 feet distance and the patient sitting upright, 40 inches or more should be used for other regions with the patient lying on the couch. It is usually impossible to obtain a good film of the upper thoracic vertebra in the lateral position and for that region reliance has to be placed on the stereoscopic view in the antero posterior position. In the case of a patient with sloping shoulders it may be possible to include the first and second thoracic bodies in the lateral view for the cervical region if the shoulders are forcibly depressed. Another method is to force the shoulders forwards by traction on the arms while the patient is standing. The routine advised by *Camp* is satisfactory. He takes antero posterior and lateral films and supplements these with stereoscopic views at any suspicious level. In addition, he usually takes right and left oblique views to show the intervertebral foramina. The openings of the intervertebral foramina lie in the oblique plane in the

cervical region but tend to face more laterally in the thoracic region while in the lumbar spine the openings are almost completely in the lateral plane

### ANATOMY

The spinal cord is continuous above with the medulla oblongata at the upper border of the atlas. In the third month of foetal life it has the same length as the vertebral column but thereafter the vertebral column grows in length more rapidly than the spinal cord. In the adult the spinal cord ends at the lower border of the first or upper border of the second lumbar vertebra. Most spinal segments thus lie at a higher level than the corresponding vertebrae.

There are eight pairs of cervical nerves. The first pair issue from the spinal canal between the occipital bone and the first cervical vertebra. The eighth pair between the seventh cervical and first thoracic vertebra. At lower levels the nerves correspond exactly in number with the vertebrae and issue from the spinal canal below the corresponding vertebra. The upper cervical nerves pass transversely from the cord to the intervertebral foramina. At lower levels the spinal nerves pass obliquely downwards in the spinal canal to reach their foramina of exit. The lumbar and sacral nerves pass almost vertically downwards forming bundles of nerves which from their appearance and length are collectively termed the cauda equina. The nerves that supply the limbs are much larger than the rest of the spinal nerves and where they are the spinal cord is larger than elsewhere. The cervical enlargement extends from the third cervical to the second thoracic vertebra. The lumbar enlargement from the ninth to the twelfth thoracic vertebra.

### RADIOLOGICAL FEATURES

In radiological examination of the spinal column the following features should be systematically observed both in antero-posterior and lateral planes.

- (1) The alignment of the vertebral column
- (2) The appearance of the vertebral bodies. their shape and outline. their density and the intervertebral distance
- (3) The size and shape of the pedicles
- (4) The intervertebral foramina
- (5) The laminae and spinous processes
- (6) The transverse processes. the adjacent parts of the ribs. normal and abnormal paravertebral shadows

Destruction of vertebral bodies is easily recognised. it is common in primary vertebral disease (Chapters XXVIII and XLVI) whereas erosion from tumours arising within the spinal canal is rare and affects only the posterior aspect of the bodies. The intervertebral discs resist erosion by tumour. The intervertebral distance alters in certain lesions of the vertebral bodies such as tuberculosis but it may also be affected in lesions of the disc itself. Thus Hampton

and Robinson report reduction in the intervertebral distance in 25 per cent of their cases of herniation of the intervertebral disc in the lower lumbar spine

The most important information is often obtained from study of the pedicles. The antero-posterior projection shows well the breadth of the pedicles and alterations of them produced by tumours within the spinal canal (Fig 170). The lateral projection shows the pedicles separating the intervertebral foramina and the effects of tumours within the foramina (Fig 171).

In the cervical region the shadow of the pedicle is circular<sup>1</sup> in the thoracic and lumbar regions it is oval. Throughout the vertebral column the medial aspect of the pedicles is straight or



FIG 171 —Large dumb-bell neurofibroma arising on the fifth cervical nerve root and causing enlargement and erosion of the intervertebral foramen between the third and fourth bodies. (Case No. L.H. 10234 1939)

<sup>1</sup> In the cervical region the limits of the shadow cast by the pedicles in the antero-posterior projection are sometimes difficult to assess in spite of good films

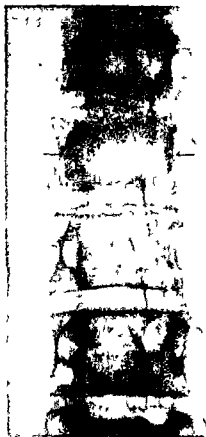


FIG 170 —Narrowing of pedicles of second lumbar vertebra and, to a lesser extent of the right pedicles of third and fourth lumbar vertebra by a large dermoid cyst of the cauda equina. A girl, aged 18. (Case No. 2410)

slightly convex except in children, in whom the pedicles in the upper part of the spine may be concave

Valuable information may sometimes be obtained from measuring the distance between the inner aspects of the right and left pedicles of each vertebra. This interpedicular distance is greatest at the cauda equina and at the cervical and lumbar enlargements, and least in the mid thoracic area,

these variations in size at different spinal levels are fairly constant from subject to subject and are moreover gradual. Charts have been produced showing the average and extremes of measurements of interpedicular distance of a large number of normal subjects. In the upper cervical spine the measurement is about 3 cm. The distance rises to 3.3 cm at the fifth cervical body and then steadily decreases until it reaches 2 cm at the fifth thoracic body. It remains at approximately 2 cm until it starts to increase again at the tenth thoracic body and increases to 3.5–3.9 cm at the fifth lumbar vertebra. Any change such as an increase where a decrease should normally be found especially an abrupt increase would indicate an expanding lesion. It must be stated however that tumours in the spinal canal do not invariably produce significant changes in the interpedicular distance.

### Expanding Lesions of the Vertebrae

The expanding lesions of the vertebrae—tumours, tuberculosis, inflammatory diseases and so forth—are considered in detail later in this volume (Chapters XXVIII and XLVI). We are concerned here only with such vertebral lesions as produce spinal compression.

*Hæmangioma* though common is a post mortem finding according to

*Topfer* rarely produces symptoms. Its radiological diagnosis is nevertheless of considerable importance because the available experience suggests that operation is highly dangerous to life whereas radiotherapy is effective in relieving symptoms (*Lathras* and *Ramage*). The disease usually affects one vertebra only or occasionally two at a distance from one another. There is diminished density of the vertebral body with uneven absorption of all trabeculae except those running in the vertical plane. This gives the appearance of parallel vertical striae in a semi-translucent body. No other disease gives quite the same appearance of a porous vertebra that does not collapse.

Giant cell tumour (osteogenetic myeloma) also occurs. The picture is usually one of expansion with coarse trabeculae running across a translucent cystic matrix. In these cases the vertebra usually collapses early and compresses



FIG 177 Eroo of right half of lamina of thoracic vertebra (the central vertebra in the illustration) by para vertebral giant cell tumour. Case No 1793

the cord. At times the tumour may arise in the lamina or adjacent soft tissues destroying the bone and pressing on the cord (Fig. 172).

Of the malignant diseases *sarcoma* is rare and as elsewhere it usually causes a large mass of soft tissue with comparatively little bone destruction. *Secondary carcinoma* is by far the commonest malignant tumour of the spine. In these cases pain in the back or along the spinal roots followed by spinal compression may be the first symptoms of illness and the true nature of the disease is only suspected after radiographic examination.

Myelomatosis (plasma celled myeloma) may also cause spinal compression or collapse before any other symptoms. Other bony lesions which may cause pressure on the cord include tuberculosis of the spine (Pott's disease), fracture, Kummell's disease, Hodgkin's lymphogranuloma, osteitis deformans, osteitis fibrosa, hydatid cyst, chordoma, eccchondrosis and protrusion of the intervertebral disc. This last condition usually produces no changes in simple radiograms.

### Paravertebral Tumours

Tumours alongside the spinal column occasionally produce spinal compression. The majority of them are neurofibromas arising in the spinal nerve roots as they leave the spinal canal. These tumours grow mainly in the paravertebral tissues but often extend through the intervertebral foramen and are known as hourglass tumours. They occur at any spinal level but are most common in the thoracic region. The mediastinal part of the tumours shows well in radiograms. In all cases the intervertebral foramen can be shown to be dilated. At times the adjacent ribs are seen to be forced apart.

Other paravertebral tumours occur such as chondrosarcoma and giant cell tumour. These may extend into the spinal canal by erosion of the laminae. The special importance of radiology in paravertebral tumours is that the part of the tumour that lies outside the spinal canal usually gives rise to no symptoms and is only detected by radiography.

### Tumours arising within the Spinal Canal

Tumours arising within the spinal canal may be (a) extradural (b) extramedullary (i.e. within the dura but outside the spinal cord) and (c) intramedullary (i.e. within the spinal cord). The most common variety of intra-spinal tumour is the extramedullary tumour. *Dyke* states the relative frequencies as extradural 20 per cent, extramedullary, 60 per cent and intramedullary 20 per cent.

The radiological diagnosis of spinal cord tumours depends chiefly on (i) deformity of pedicles of the vertebrae, (ii) increase in interpedicular distance and (iii) destruction of part of a vertebral body or lamina. Rarely the shadow of a tumour may be seen projecting through the intervertebral

foramen into the surrounding soft tissue as already described and more rarely calcification may be demonstrable in a tumour especially in meningiomas



FIG 173 —End view of the posterior aspect of the bodies of the lumbar vertebrae by a large cauda equina tumour in a woman aged 40 years. The intervertebral discs are resistant and so the erosion is at least at the upper and lower margin of the bodies. Case No. L.H. 40041 1939

especially the variety known as ependymoma) neurofibroma dermoid teratoma or lipoma. Whatever its structure this tumour is usually a large ovoid tumour of some 2 inches length the so called giant tumour of the cauda

Deformity of the pedicle is brought about by pressure its medial surface becomes concave where normally it is convex or flat. If the pedicles of the affected vertebra are compared with each other and with those of the vertebrae above and below encroachments can be seen (Fig 170)

Extradural tumours of soft tissue include meningioma neurofibroma lymphogranuloma extradural cyst and angiomatous malformation. In the lumbar region and occasionally in other parts of the spinal canal protrusion of the intervertebral disc into the spinal canal occurs.

Extramedullary tumours consist in approximately equal proportions of neurofibromas of the nerve roots and meningiomas attached to the dura. They are usually less than 1 inch long. As they grow they embed themselves in the spinal cord. A few of the meningiomas are calcified but the majority of extramedullary tumours can only be displayed radiologically by changes in the pedicles and these are by no means invariable. Extramedullary tumours both meningiomas and neurofibromas may be occasionally multiple particularly in the subjects of von Recklinghausen's neurofibromatosis. Simple epithelial and arachnoid cysts are also sometimes found.

A special variety of extramedullary tumour is the tumour of the cauda equina which may be a glioma (es

equina (Fig 173) It may produce considerable distortion of the pedicles and widening of the interpedicular distance and occasionally erosion of the vertebral bodies

Intramedullary tumours are nearly all cystic and solid gliomas though dermoids lipomas and hæmangioblastomas also occur These tumours may be prolonged through several segments of the spinal cord The clinical picture is sometimes and the lipiodol picture is often distinctive, but it may be impossible to diagnose the intramedullary position of the tumour until operation All intramedullary tumours are capable of causing erosion of the overlying bone but seldom do so and the simple radiograms thus usually show no positive signs

### INTRATHECAL LIPIODOL

Lipiodol in oil of poppy containing about 49 per cent of iodine was first used for the diagnosis of spinal tumours by Sicard and Forestier in 1921 It is opaque to X rays and heavier than cerebrospinal fluid its passage down the spinal theca can thus be watched on the screen and any obstruction confirmed by radiography Its chief use therefore is in diagnosing the presence and exact level of an obstruction and it may be employed in investigating cases of block following collapse of a vertebra due to disease or trauma dislocation of the spine and adhesive arachnoiditis as well as cases of spinal tumour Small encroachments on the spinal canal such as those caused by protrusion of the intervertebral disc can also be detected though they do not cause an obstruction but only give rise to a constant filling defect

The great virtue of lipiodol is that it is almost free from irritating effects upon the meninges nerve roots and the spinal cord It is not absorbed from the spinal theca but becomes encysted in the sacral cul de sac and during a period of years extends outwards from the spinal canal for a variable distance along the lumbar and sacral nerve roots In the majority of patients its presence and retention in the spinal canal produce a cellular reaction of variable intensity, but this is not followed by any symptoms or neurological signs On a few occasions however it is reported to have set up adhesive arachnoiditis

### Indications

In consequence of this danger even though it be slight intraspinal lipiodol should not be used indiscriminately Its greatest value is in identifying the exact upper or lower limit of a spinal tumour whose presence is certain as a result of clinical study and spinal manometry In such cases the best neurologist may often be one or two spinal segments out of his estimation of the level of the tumour and the surgeon may be one or two vertebrae out of his surface markings, especially in fat people

To appreciate the indications for intrathecal lipiodol the radiologist should be conversant with spinal manometry and the Queckenstedt test



Lumbar puncture can be done with safety through any of the lumbar inter spaces except the first since the spinal cord ends at the lower border of the first or upper border of the second lumbar vertebra. The resting spinal pressure is 150 mm of  $H_2O$  but pressures between 70 and 180 mm of  $H_2O$  are within limits of normal. The fluid in the manometer shows slight fluctuation with respiration and it also rises if the abdominal veins are compressed by strong compression of the abdominal wall. These fluctuations indicate that the point of the needle in the subarachnoid space is free from obstruction.

Queckenstedt's test consists in noting the effect of compression of the internal jugular veins in the neck upon the pressure of the spinal manometer. In an earlier chapter an explanation has been given of the effect of venous obstruction upon intracranial pressure. Jugular compression causes a rise of intracranial pressure, cerebrospinal fluid is in consequence displaced down into the spinal canal and there is a rise of pressure in the spinal manometer. Compression of the jugular veins for ten seconds will cause a prompt rise of the spinal pressure from 150 to 400 mm of  $H_2O$  and an equally prompt fall occurs when the pressure on the neck is released. This is the normal response. But if there is any obstruction in the spinal canal above the needle point the rise of pressure on jugular compression will not be conveyed down to the lumbar region and in consequence the fluid level in the manometer will not rise or may only rise slowly and incompletely or having risen it will not fall. These are the positive results obtained by Queckenstedt's test and always provided the jugular veins and lateral sinuses are patent and the skull is intact they indicate an obstruction in the spinal canal from tumour, arachnoidal adhesions, bony displacement and so forth.

It is obvious that if the tumour lies in the lower lumbar and sacral part of the spinal canal Queckenstedt's test may be negative even though the needle is introduced through the lowest available space. Experience shows that at the upper end of the spinal canal also tumours may exist and produce serious symptoms of spinal compression with little or no obstruction on Queckenstedt's test. With the exception of these tumours at the upper and lower ends of the canal spinal tumours tend to produce a blockage on Queckenstedt's test.

Lipiodol should not be employed for investigation of a suspected case of spinal tumour until full clinical studies and spinal manometry have been carried out. In most cases if Queckenstedt's test is normal it is useless to employ lipiodol for it will usually pass quickly to the sacral cul de sac without revealing the tumour or from being temporarily held up at one point it may induce the belief that there is a tumour where none exists. There are however some special indications for the use of lipiodol in cases where there is no spinal block shown by manometry, thus lipiodol is used to display protrusion of the intervertebral disc and to detect high cervical tumours.

The use of lipiodol in spinal tumour may occasionally be followed within a few hours by an increase of the paralysis, retention of urine and other signs

of compression but no permanent harm is done if the tumour is removed within the next twenty four hours. The same type of aggravation of signs may follow simple lumbar puncture, and from the same cause, namely a shift of the tumour in relation to the spinal cord. At operations after injection of lipiodol the oil obstructed in the region of the tumour, is for the most part removed.

### Technique

Lipiodol is injected into the subarachnoid space either through the cisterna magna or by lumbar puncture. A tilting table is essential for thorough examination though an incomplete examination by the cisternal route can be made with the patient sitting up. A detailed description of the technique is given by *Camp* and by *Worth*.

**Cisternal Injection**—For cisternal puncture the back of the head is shaved up to the external occipital protuberance and cleaned with antiseptic, a wheel is then made with 1 per cent novocaine at a point in the middle line on a level with the spine of the axis. With the patient's head slightly flexed in order to separate the posterior arch of the atlas from the base of the skull a lumbar-puncture needle is introduced in a forward and upward direction towards the root of the nose so that it will strike against the base of the skull behind and above the foramen magnum. It is then partly withdrawn and is advanced once more in a more horizontal direction so as to pass into the theca below the posterior edge of the foramen magnum. The occipito atlantal ligament is so tough that a distinct increase of resistance is always felt as the point of the needle passes through it. Care should be taken that the needle only just enters the cisterna magna as there is danger of injuring the vertebral arteries or the medulla oblongata if the needle is advanced farther. On rare occasions cases have been reported of subarachnoid hæmorrhage following cisternal puncture which required immediate operation in order to save life. On withdrawing the stylet cerebrospinal fluid usually drips from the needle but sometimes in the sitting position the pressure in the cisterna magna is so low that fluid comes away only on aspiration.

A specimen of the cerebrospinal fluid from the cisterna magna is taken for examination and lipiodol is then injected in amounts varying from 2 to 5 c.c. The less complete the spinal block on Queckenstedt's test, the greater the amount of oil that should be used. With small amounts there is a tendency for the oil to break up into globules, and for arrest of the oil by normal structures.

With the screen the lipiodol is observed in its course down the spinal canal. Its passage will be slowest where the cord is largest, namely at the cervical and lumbar enlargements. Extreme curves in the spine will cause a delay in the rate of flow. In addition the lipiodol may be held up temporarily by spinal roots, the dentate ligaments and the septum posticum. Films are

taken whenever there is any check to the flow of the oil. Lateral as well as antero posterior projections may be necessary.

Wherever a block is found the examination should be repeated after a few hours and if necessary, next day, to exclude a partial or false check. If there is no obstruction the lipiodol drops into the sacral sac which ends at the level of the second or third sacral vertebra. On a tilting table the lipiodol can then be sent on the reverse journey, so that the whole spinal canal is again examined, but it should not be allowed to enter the skull, for there it is liable to produce headache and irritation of the posterior cranial nerves. The examination is made with the patient prone and supine. After one or two weeks the lipiodol usually becomes fixed in the sacral cul-de-sac and can no longer be manipulated for the purpose of radiographic examinations.

**Lumbar Injections.**—The lumbar route should always be used when a tumour in the region of the foramen magnum is suspected and when the lower lumbar and sacral parts of the canal are to be examined as for suspected protrusion of an intervertebral disc. It has indeed many advantages over the cisternal method for nearly all cases since it does not require shaving of hair, and the risks of lumbar puncture are even smaller than those of cisternal puncture. It shows also the lower limit of the tumour, and many surgeons prefer to know before operation the lower rather than the upper limit of the tumour.

For the lumbar injection a tilting table is an absolute necessity. It is



FIG. 14.—Protrusion of the intervertebral disc between the fifth lumbar body and upper part of the sacrum causing a constant filling defect (marked by arrow) in a column of spinal lipiodol. Case No. 2271. 193

surprising how much downward tilt of the trunk is necessary to cause the lipiodol to overcome the normal curves of the spinal column in its passage from the lumbar to the cervical region. The lipiodol is injected by lumbar puncture with the patient lying on the tilting table. The patient is then tilted trunk and head downwards the tilt being gradually increased whenever the screen shows that the lipiodol is held up. This examination is conducted with the patient face down or on his back. If the level is judged by such structures as

the diaphragm and heart it is important to bear in mind that there is considerable distortion. All impressions from screening must be controlled by radiograms and 15 by 12 inch films should be employed. Lateral and oblique projections can be also used.

Where a block is found that cannot be overcome by 80 degrees head down wards tilt a second radiogram should be made fifteen to thirty minutes after the first to ensure that there is no false check.

For examination of the lumbo sacral sac the patient is tilted feet downwards after injection of

the lipiodol 5 c.c. of oil should be used and the

tilt should be done slowly in order to keep the oil together as a continuous shadow, and to prevent it from breaking up into globules. If search is being made for a protrusion of an intervertebral disc all that will be seen usually is a niche in the lipiodol shadow, and radiograms must be taken not only in antero posterior, postero anterior, and both lateral positions but also in oblique planes otherwise the niche produced by the protruded disc may not be disclosed (Fig 174).

Occasionally as for example when multiple spinal tumours are suspected it may be necessary to use both cisternal and lumbar injections in the same patient. According to Worth some tumours may be demonstrated by cisternal injection after lumbar injection has failed to show any lesion.

### Interpretation

Lipiodol displays the precise level of the upper or lower limit of the lesion and in a few cases indicates its nature, though for the most part the lipiodol shadows related to the



FIG 173.—Cisternal lipiodol completely blocked by an extradural cyst in the upper dorsal spine. The lower margin of the lipiodol is higher on right than the left. Woman, age 138 (Case No. L.H. 2334/1913).



FIG 174.—Lipiodol around an extradural neurofibroma of the lumbosacral spine (Case No. 1939).

various types of spinal tumour have not been as yet clearly defined. When there is a complete block the lower limit of the shadow in a case of cisternal injection has a flat or concave lower edge often with a downward spur at one point (Fig 175). This spur may even be prolonged around the lower pole of the tumour (Fig 176). Vertebral lesions and most extradural lesions tend to produce a shadow which has a transverse margin against the



FIG 177 Cisternal lipodol showing complete transverse block at level of the 11th thoracic vertebra. A case of osteosarcoma of the paraplegia. Case No. 31.

tumour. This transverse margin may be a broken line resembling the end of a bundle of faggots (Fig 177). Extradural tumours tend to produce a sharp margin concave towards the tumour. Intramedullary tumours at times show the oil breaking up into two streams of nodular outline on each side of a central space, an appearance which corresponds to a fusiform enlargement of the spinal cord (Fig 178 (a and b)). At times however oil will divide into two streams on either side of a normal spinal cord, thus interpretation of these appearances must be made with caution.

Inflammatory conditions giving rise to arachnoiditis may cause a complete block in the spinal canal, but more commonly the oil is held in droplets and globules scattered over the involved area. The droplets of oil remain stationary over a varying period. If the adhesions are severe the oil will never move and by its slightly irritant effect may even accentuate the inflammation and adhesion. It is probable that the oil lodges in pockets and therefore no physical methods such as movement or alteration of intrathecal pressure can dislodge it. If a block is complete the oil will be seen to indicate the nerve roots at the site of block by small triangular lateral projections. Occasionally the lipodol will travel quite an appreciable distance along a root, this is common if the oil lies above a lesion for some time and no operation is performed.

Coggeshall and van Storch have described a method of investigating the sacral sac by injecting air into the lumbar region after withdrawal of some fluid and then tilting the patient until the head is 30 degrees below the horizontal plane. The air rises to the sacral sac and thus the outline is radiographed.

This method of investigation is being used increasingly for examination



FIG 1-8(a)—Lipiodol above an intramedullary abscess of the spinal cord at tenth thoracic segment Case No 230<sup>1</sup>



FIG 1-8(b)—Intramedullary ependymoma of upper thoracic spinal cord Case No 235<sup>1</sup>

of the whole spinal cord using oxygen or air instead of lipiodol and with improvement of technique and increase of detail in the radiograph it is possible that it may replace the oil injections. It is possible to detect many tumours by this means at the present time. Lindgren advocates the use of oxygen in preference to air and states that it causes less inconvenience and reaction to the patient.

<sup>1</sup> The piece of silver wire seen in the radiogram is used to correlate the skin level with the intraspinal level for the purposes of operation.

## BIBLIOGRAPHY

- CAMP, J. D. Multiple Tumours within the Spinal Canal. *Amer Journ Roentgenol* 1936 XXXVI 70.
- CLOWARD, R. and BUCK, P. C. Spinal Extradural Cyst and Kyphosis Dorsalis Juvenilis. *Amer Journ Roentgenol* 1937 XXXVIII 681.
- COGGERSHALL, M. C. and VAN STORCH, T. J. C. Diagnostic Value of Myelographic Studies of the Caudal Dural Sac. *Arch Neurol and Psychiat* 1934 XVI 611.
- ELSBERG, C. A. Tumours of the Spinal Cord. 1925. New York.
- HAMPTON, AUBREY O. and ROBINSON, J. The Roentgenographic Demonstration of Rupture of the Intervertebral Disc into the Spinal Canal after Injection of Lipiodol. *Amer Journ of Roentgenol* 1936 XXXVI 78.
- REUER, G. The so-called Hour glass Tumours of the Spine. *Arch of Surg* 1929, XVIII 935.

- LINDGREN, E., 'Über Skeletveränderungen bei Rückenmarkstumoren,' *Neurologisch*, 1937, X, 240, "On the Diagnosis of Tumours of the Spinal Cord by the Aid of Gas Myelography," *Acta Chir Scand*, 1939, LXXXII, 303
- LOVE, J. GRAPTON, and CAMP, J. D., "Root Pain Resulting from Intraspinal Protrusion of Intervertebral Discs," *Journ Bone and Joint Surg*, 1937, XIX, 776
- MIXTER, W. J., and BARR, J. R., "Rupture of the Intervertebral Disc with Involvement of the Spinal Canal," *New England Journ Med*, 1934, CCXI, 210
- NATTRASS, F. J., and RAMAGE, D., Hæmangioma of Vertebra A Cause of Compression of the Cord, *Journ of Neurol and Psychopathology*, 1932, XII, 231
- ODIN, M., and RUNSTROM, G., 'Iodised Oils,' *Acta Radiol*, 1928, Suppl VII
- SICARD, J. A., and FORESTIER, J. E., 'Méthod Générale d'Exploration Radiologique par l'Huile Iodée (Lipiodol)' *Bull et Mem de la Soc Med des Hôp de Paris*, 1922, XLVI, 463 Radiological Exploration with Iodized Oil," *Brit Journ of Radiol*, 1926, XXI, 239
- TOFFER, D., 'Ueber ein infiltrierend wachsendes Hamangiom der Haut und multiple Kapillarektasien der Haut und inneren Organe Zur Kenntnis der Wirbelangiome,' *Frankfurt Zeit f Path*, 1928, XXXVI, 337
- YOUNG, B. R., and SCOTT, M., "Air Myelography," *Amer Journ Roentgenol* 938, XXIX, 187

*PART TWO*

THE ACCESSORY NASAL SINUSES, LABYRINTH AND  
MASTOID PROCESS

BY

H K GRAHAM HODGSON, *C V O*, MB BS FRCP, FFR DMR



## PART TWO

# THE ACCESSORY NASAL SINUSES, LABYRINTH, AND MASTOID PROCESS

## CHAPTER VII

### ANATOMY OF THE ACCESSORY NASAL SINUSES

ALTHOUGH A thorough knowledge of the anatomy of each and every part of the body is an essential concomitant of the radiologist's equipment if he is to diagnose accurately, it is especially necessary in the case of the accessory nasal sinuses. In the first place, the normal varies within very wide limits, indeed, the shape, size, and developmental abnormalities of the sinuses of different individuals vary so greatly that no two persons' sinuses are alike. So true is this, that, given good radiograms taken by correct standard technique, an experienced radiologist could pick out with certainty the radiograms of one individual from amongst a thousand others, a fact which incidentally might be, under certain circumstances, of considerable forensic value. In the second place, the radiologist's examination is frequently preliminary to surgical treatment and here the surgeon is operating on structures which he either cannot see or of which he can only obtain a very imperfect view. How essential it is, therefore, that the X ray information on which he depends for his guidance should be provided by one whose knowledge of the anatomy of the part is equal to his own. In addition to a thorough knowledge of the anatomy, pathology, radiographic technique, and interpretation of radiograms of the accessory nasal sinuses—subjects which are dealt with here—the radiologist should also acquire a working knowledge of the procedure of the surgery of these parts so that he may understand the requirements of, and fully co-operate with, his colleague, the surgeon.

### THE MAXILLARY SINUSES

When viewed in the postero anterior plane, the maxillary sinuses are pyramidal in shape, with the apices directed downwards. In the lateral plane they are quadrilateral, with rounded corners. In the vertical view they are pyramidal, with the apices directed backwards.

The antrum is situated in the body of the maxilla. Above, it is bounded by the floor of the orbit, below by the molar and bicuspid teeth of the upper jaw, mesially it is bounded by the lateral wall of the nose, anteriorly and

laterally by the facial wall of the maxilla and posteriorly by the posterior wall of the body of the maxilla separating it from the pterygo maxillary fossa

The antrum like all the accessory nasal sinuses is lined with ciliated epithelium and the ostium or opening is situated in the upper part of the nasal wall. Consequently when the body is in the erect posture drainage of the sinus is entirely dependent on ciliary action.

The antra are the most symmetrical of the accessory nasal sinuses and are the least subject to developmental variations. The most common variation is in the level of the antral floor which may vary from 1 cm. below to 1 cm. above the nasal floor. The position of the antral floor is of surgical importance where an intranasal puncture is performed and any abnormality in its position should be noted in the X ray report.

The antra are usually of equal size but occasionally one may be small and poorly developed the bony wall being correspondingly thicker. Such a sinus will naturally be more opaque to X rays than its fellow of the opposite side and one must be careful not to interpret this opacity as pathological.

Particular attention should be paid to the floor as early pathological changes of an inflammatory nature are most marked here. The relationship of the teeth of the upper jaw to the antral floor should also be noted. When the antral floor is prolonged downwards the roots of the molar and bicuspid teeth appear to project into the antrum. Stereoscopic films will usually show them to lie in the outer wall of the sinus. They do however occasionally project into the antrum and are then covered by the antral mucosa. It is in these cases that an apical abscess may most readily cause infection of the antrum and extraction of one of these teeth may cause a breach in the antral wall.

The internal walls of the antra are usually smooth but occasionally partial septa may project into the cavity and in very rare cases a complete septum may divide the sinus into two parts. It is important to recognise the presence of such a septum as in the event of infection the surgeon may puncture the uninfected part and find a clean sinus whereas the other part may be filled with pus. Where the radiograms suggest the possibility of such a condition the introduction of lipiodol through the ostium will establish the diagnosis. The lower border of the middle turbinate bone which in the lateral view is seen to cross the antrum from behind upwards and forwards must not be mistaken for a septum.

In infants the antrum cannot be distinguished radiographically as a definite cavity until the end of the first year and until the twelfth year the floor is largely concealed by unerupted teeth.

### THE FRONTAL SINUSES

As their name indicates these sinuses are situated in the frontal bone—in the horizontal and vertical portions. Below they are bounded by a varying

area of the roof of the orbit and the ethmoid labyrinth. Above they extend into the vertical portion of the frontal bone.

The frontal sinuses are frequently asymmetrical and they vary much in size and shape. The larger the frontal sinuses are the greater variations they show in shape. An average sized fully developed frontal sinus extends from the midline to the supra-orbital notch. At the fifth year they are about the size of a threepenny bit and they gradually increase in size until they reach full development at about the eighteenth year.

The frontal sinuses may vary in size from complete absence of one or both sinuses to extension throughout almost the whole frontal bone in the vertical direction and horizontally and backwards to the wings of the sphenoids. Congenital absence of one frontal sinus is comparatively common occurring in one in twenty persons but absence of both is much more rare and is usually associated with a persistent metopic suture. In cases where the vertical portions of one or both frontal sinuses are absent the stereoscopic lateral films should be carefully studied to determine whether the horizontal portions are present as absence of the former is more common than absence of the latter.

The anterior wall may be irregular in thickness with consequent irregularity in density on the film which may be interpreted by the inexperienced as pathological. On the other hand the posterior wall is smooth and of a uniform thickness throughout.

The frontal inter sinus septum may be centrally placed or deviated to one side or the other. Occasionally it may be absent. Partial septa are frequently seen in large frontal sinuses projecting from the anterior wall.

When surgical procedure is contemplated it is important for the surgeon to know the size and shape of the frontal sinuses and the presence or absence of ethmoid cells in the immediate neighbourhood of the fronto-nasal duct. When an external operation is to be performed it is obviously important to know the level of the floor and also the antero-posterior depth of the sinus. If the anterior wall of the sinus is removed at operation and this antero-posterior depth is excessive unsightly hollowing or flattening of the forehead will result.

The fronto-nasal duct usually communicates directly with the superior meatus or the hiatus semilunaris but occasionally it may open into a misplaced ethmoidal cell which in turn opens into the superior meatus.

Aberrant ethmoid cells not infrequently complicate operation on and drainage of the frontal sinuses and it is important that the radiologist should recognise their presence and inform the surgeon of their location. For this purpose stereoscopic lateral views are the most valuable. Such cells may project into the floor of the frontal sinus may be in close contiguity with the fronto-nasal duct or a cell may be situated in the agger nasi anterior to the duct. It is obvious that disease in these cells may infect the neighbouring

frontal sinus, and that under such circumstances it would be useless to operate on the frontal sinus without dealing, at the same time with these cells

### THE ETHMOIDAL CELLS

The ethmoidal labyrinths are bounded above by the floor of the anterior fossa and below by the hiatus semilunaris. Mesially, each ethmoid labyrinth is bounded by the middle meatus and the nasal septum, and, laterally by the orbital plate of the ethmoid.

Of all the accessory nasal sinuses, the ethmoid labyrinth is subject to the greatest developmental variations. It is therefore essential for the radiologist to have an accurate and wide knowledge of the anatomy and these developmental variations of the ethmoid labyrinth for not infrequently the detection of an aberrant and infected ethmoid cell may supply the key, unobtainable except by radiology, to the patient's symptoms.

Misplaced ethmoid cells are found far outside their own anatomical boundaries, and the careful radiologist should always be on the look out for these 'Bedouins' of the accessory nasal sinuses. In searching for these wandering ethmoid cells stereoscopic lateral views are very useful in addition to the standard oblique views.

The ethmoid cells vary in size and number and lie on each side of the midline of the nose comprising the two ethmoid labyrinths. Each labyrinth may in rare cases consist of only three or four large cells but usually a large number of smaller cells are present the usual number being twelve to sixteen. The cells are differentiated into anterior and posterior groups. The posterior ethmoid cells are usually larger than the anterior cells. Each cell has a separate ostium. The ostia of the anterior cells open above or into the hiatus semilunaris those of the posterior cells into the superior meatus and the sphenoidal recess but this is an anatomical distinction which cannot be distinguished in a radiogram.

The superior border of the ethmoidal capsule runs backwards from the frontal sinus to the sphenoidal sinus. The posterior border is in contact with the sphenoidal sinus. The lower border of the capsule extends forwards and curves into the anterior border which runs upwards and forwards to join the superior border in a rounded eminence, known as the ethmoidal bulla following the line of the hiatus semilunaris. Cells situated in front of the fronto-nasal ducts and behind the frontal processes of the maxillæ are known as agger nasi cells.

Cells are not infrequently found outside the ethmoid labyrinth in various situations. It is obvious how extremely important it is for the radiologist to recognise a cell of this type and report on its presence to his surgical colleague. The fronto-ethmoidal cell is the type of aberrant ethmoid cell most commonly met with. It is situated between the supra-orbital margin and the anterior

part of the floor of the frontal sinus. It is usually about the size of a pea, but it may occasionally extend well up into the frontal sinus, or backwards over the orbit. In the latter case it is sometimes difficult to distinguish an aberrant cell of this type from a large horizontal extension of the frontal sinus, but the ethmoid cell usually is subdivided by septa or partial septa.

Again, aberrant cells are not infrequently found in the body of sphenoid, either alongside or under the sphenoidal sinus; occasionally they may lie over the sphenoidal sinus. The confusion of shadows, caused by the projection of such cells on the shadow of the sphenoidal sinuses, at first makes interpretation difficult, but stereoscopic lateral views, in addition to vertico mental views, should lead the radiologist to the solution of the problem. Sometimes, however, the introduction of lipiodol is necessary to clear up the position.

Ethmoid cells are sometimes found in the pterygoid processes. The fronto nasal duct may open into an ethmoid cell, and the frontal sinus gain exit to the nose through the ostium of the cells.

The position of the bulla ethmoidalis varies to a fairly wide extent and it may occasionally project so far forward as to narrow or distort the fronto nasal duct; it may touch the middle turbinate or push it against the nasal septum.

Lastly, ethmoid cells are frequently found between the floor of the orbit and the roof of the antrum, and may occasionally extend for some distance down the facial wall of the antrum.

From the above description it will be seen how essential it is for the radiologist to have a very complete knowledge, not only of the normal anatomy of the ethmoid cells, but also of their many developmental abnormalities. An operation may be rendered useless by failure to open an infected aberrant ethmoid cell, which often can be detected only by radiology.

### THE SPHENOIDAL SINUSES

The sphenoidal sinuses are usually not visible radiologically until about the beginning of the third year. Pneumatisation extends from before backwards and reaches the dorsum sellae about the tenth year.

Developmental absence of a sphenoidal sinus is extremely rare. They are usually asymmetrical and are situated in the body of the sphenoid bone. They vary considerably in size and shape, but their floors are always parallel with the floor of the pituitary fossa. Their cavities sometimes extend upwards into the dorsum sellae. They are divided by a septum, which is frequently not median, and varies in thickness. Accessory pockets or loculi may project from the lateral or posterior wall of the sinuses. Again a para sphenoidal ethmoid cell may open into one of them. The sphenoidal sinuses open into the sphenothmoidal recess, the opening being high up in the anterior walls of the sinuses.

As in the other sinuses, so in these, where individual variations are so wide and so frequent close study of the anatomy of each case is essential, and

attention should be drawn by the radiologist to any point which might be of help to the surgeon who has to depend almost entirely on the radiologist for warning of any peculiarity of the anatomical structure in the posterior group of sinuses

Finally the writer would recommend the student to study stereoscopic radiograms of the sinuses carefully and persistently until he can identify every shadow

## CHAPTER VIII

### TECHNIQUE OF EXAMINATION

IN X-RAYING the skull one is X-raying the most complicated bony structure in the body—one is throwing multiple shadows widely differing in distance, size and density on to the single plane of the X-ray film. A small difference in the angle of incidence of the rays in the exact point on the skull over which they are centred or in the positioning of the patient's skull will swing the shadows of those structures which are a few inches away from the film through a wide arc and cause a big variation in their relative positions to the shadow of structures closer to the film. Hence the absolute necessity for taking the radiograms at constant and standardised angles in every case irrespective of individual differences in shape of the patient's skull for exact centring and angulation of the tube and for taking several different views from standardised angles. In the majority of text books dealing with this subject one finds only three views mentioned—a lateral view and two other views described respectively as the nose chin and nose forehead position. These are not in



FIG. 179—Nose chin position



FIG. 180—Standard position

fact, exactly defined ' positions at all and the multiple shadows in the resulting radiograms depend for their situations relative to one another on not more exact or scientific a standard than the widely varying facial contours of individual patients. It is hopeless to expect accurate X ray findings from such inaccurately taken X ray films.

For example Figs 179 and 180 are two radiograms of the same patient. Fig 179 was taken in the nose chin position and it will be seen that the lower parts of both antra are concealed by the shadows of the petrous bones, this being brought about by the patient having a long chin and a short nose. In this radiogram nothing abnormal can be detected. Yet Fig 180 taken a few minutes later at the correct angle throws the petrous bones clear of the antra and shows the right antrum to be half filled with pus.

This is only one illustration of the impossibility of demonstrating pathological conditions in the accessory nasal sinuses by X rays with any degree of accuracy if the radiographic technique employed is of a haphazard character. Given the use of correct radiographic technique the findings in the X ray investigation of the accessory nasal sinuses are as uniformly accurate as in the case of any other part of the body—if not more so. The first point to be noted therefore is that one cannot over emphasise the absolute necessity for accurate and standardised technique in the radiology of the accessory nasal sinuses.

### IMPORTANCE OF ERECT POSTURE

In the days when X ray apparatus gave only a small output and before the production of double-coated films and of intensifying screens the exposures were very long and the only position in which the patient could keep reasonably still was either the prone or the supine. In spite of the vast improvements in power and speed that have reduced the exposure to at the most a matter of six or eight seconds by far the greater number of X ray examinations—with the exception of the chest and stomach—are carried out in the recumbent position. In the majority of instances there is no disadvantage in such a position but it is definitely contraindicated when the part to be X rayed is an air containing cavity which may be abnormally opaque to X rays as a result of pathological changes in the mucosa or of pus. If one wishes to examine the contents of a glass jar and to ascertain whether its content is solid or fluid one does not do so with the jar lying on its side as that would not give one the required information. One naturally holds it upright on a level with one's eyes so that the light passes horizontally through the material to one's eye. In this position one can examine the structure of the material see the upper border and ascertain whether it is fluid or not by tilting the jar from side to side. If it is fluid this upper border will remain horizontal whatever the position of the bottle. The same thing applies to the sinuses if they are X rayed.



in the upright position. If they are X rayed in the prone position gross pathological changes may easily escape detection.

Fig 181, for instance, shows no abnormality beyond a slight degree of cloudiness in the antrum. This radiogram was taken in the prone position. Fig 182 is a radiogram of the same patient but taken in the erect posture a few minutes later, which shows a collection of pus in both antra. What happens is that in the prone position the fluid is spread out in a thin film over the antral wall and when the amount is small it is insufficient to cast an appreciable shadow. In the erect position, however, the fluid is all collected into a pool in the lower part of the sinus and is at once obvious.

**Normal Transradiancy**—Another possible source of error is the widely held assumption that the radiological detection of sinus infection depends on the



FIG 181.—Prone position



FIG 182.—Erect position

observation that one sinus is more opaque than its fellow of the opposite side. One has only to consider the matter for a moment to appreciate how misleading radiological evidence based on this fallacy might often be. For, on the one hand, a sinus may be small and poorly developed with thick bony walls and consequent small air content which though non pathological is opaque when compared with the opposite normally developed sinus. On the other hand, a pair of sinuses may often contain an equal amount of pus or thickened lining membrane and since both will allow an equal amount of rays to pass they would both on this basis of diagnosis be considered radiologically normal. The only safe and reasonable basis for radiological diagnosis is the radiologist's acquaintance with the normal degree of transradiancy which each sinus should possess according to its size and air content. This necessitates a thorough knowledge of the standard normal and obviously can only be gained by taking the radiograms by a standardised technique.

## STANDARD TECHNIQUE

In order to put into practice the technical procedure outlined above it was first of all necessary to design a special piece of apparatus for this purpose

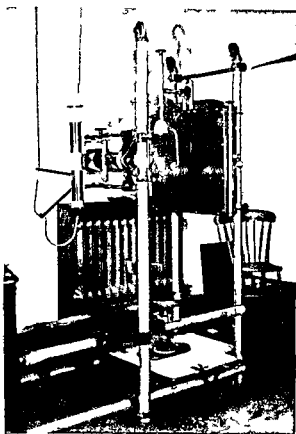


FIG. 183. Universal radiographic stand.

Fig. 183 illustrates the stand the writer designed in 1929 and except for minor alterations such as the introduction of the shock proof tube the general design of such stands remains the same to day. The head clamp is of the lazy tong type and can be rotated the angle of rotation being measured on a calibrated scale. It is attached to the Potter Bucky diaphragm which is counterbalanced for raising or lowering. Some radiologists prefer to use a long cone and dispense with the Potter Bucky diaphragm and this certainly makes the apparatus considerably less expensive. The writer prefers the radiograms taken with a Potter Bucky and also the Potter Bucky makes the stand of much greater value for the radiology of other parts of the body. The seat can be moved backwards forwards

transversely and up and down. The tube can also be moved backward and forwards and can be tilted through a measured angle.

Although at first called a 'Sinus Stand' it is so useful for X raying other parts of the body such as the cervical spine shoulder pregnancies teeth etc. that the writer thinks the term Universal Radiographic Stand better. Its initial expense therefore is offset by its utility being wider than in its original intention.

The six standard views which the writer uses are all taken in the erect position and at definite and accurately measured angles for every patient irrespective of any difference in facial contour. They are as follows:

(1) Occipito frontal View (Fig. 184) —The head is grasped by the head clamp in the bitemporal diameter and the head clamp is adjusted so that the

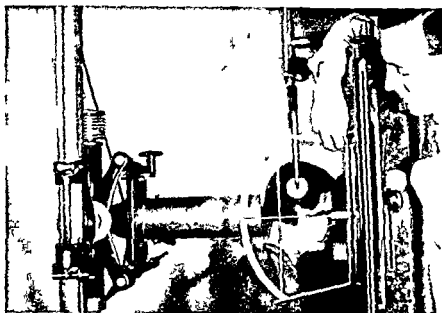


FIG. 184.—Position for occipito frontal view

antero posterior diameter of the skull is perpendicular to the film (the angle being read off on the dial). An imaginary line is taken joining the external auditory meatus and outer canthus of the eye which for brevity may be called the *orbito meatal line*. This line is so adjusted by means of a protractor that it is perpendicular to the film. The tube is centred half an inch below the external occipital protuberance and the exposure made. A typical result is

shown in Fig. 185. The petrous bones will be seen to be thrown well above the antra in this position. The principal use of this view, however, is not to show the antra, but to show whether there is any abnormal opacity of the ethmoidal labyrinth or of the sphenoidal sinuses. On either side of the perpendicular plate of the ethmoid two air spaces are seen. These air spaces in this position consist of the superimposed shadows of the anterior and posterior ethmoidal cells and the sphenoidal sinuses from before backward and normally they are clear.

(2) *Occipito mental View* (Figs 186 and 187).—In this view the head still grasped in the bitemporal



FIG. 185.—Occipito frontal view

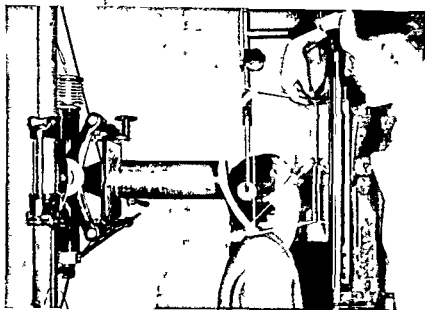


FIG 186 —Position for occipito mental view

diameter is so adjusted that the orbito meatal line subtends an angle of 45 degrees to the film this angle being open towards the feet. The resulting radiogram is shown in Fig 187. In this position the petrous bones are seen to be thrown clear below the antra thereby giving an unimpaired view of these sinuses. The anterior ethmoidal cells are thrown clear of the posterior group of sinuses and the frontal sinuses are well shown.

(3) **Vertico-mental View** (Figs 188 and 189) —In this position the patient extends the head as far back as possible on the neck. There can obviously be no standard angle through which all patients can extend the head but this is compensated for by so tilting the tube that, whatever this angle may be, the central ray is constantly at right angles to the patient's vertex.



FIG 187 —Occipito mental view

An alternative to this view and one which many patients find easier, is the mento vertical view. This is the same view, but the opposite way round, the patient placing the vertex against the Potter Bucky and the rays being centred between the horizontal ramus of the mandible.

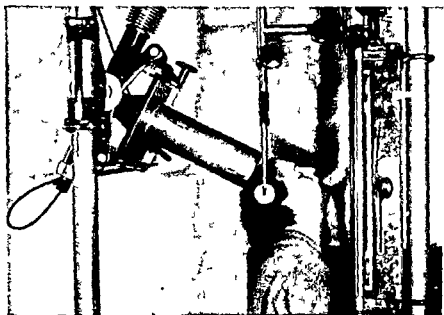


FIG. 188.—Position for vertico mental view

(4) **Right Oblique View** (Figs 190 and 191) —The head, still grasped in the clamp, is rotated through an angle of 39 degrees, the occiput moving towards the right. The chin is then raised so as to displace the mastoid downwards and so out of the way, the tube being centred just behind and above the left mastoid process. This view throws the shadows of the right posterior ethmoidal cells, and incidentally the right optic foramen, into the shadow of the right orbit.

(5) **Left Oblique View** (Fig. 192) —This is the same as the right oblique view, but in this instance the occiput is rotated through 39 degrees to the left, thereby showing the left posterior ethmoidal cells.

(6) **Lateral View** (Figs 193 and 194) —This is an ordinary lateral view of the sella turcica and accessory nasal sinuses, and is useful in cases of congenital absence of one or both frontal sinuses, and in cases in which one or other antrum is poorly developed, with thick bony walls, and consequently more or less opaque in the anterior view. The lateral view throws into relief the thick bony walls, and



FIG. 189.—Vertico mental view

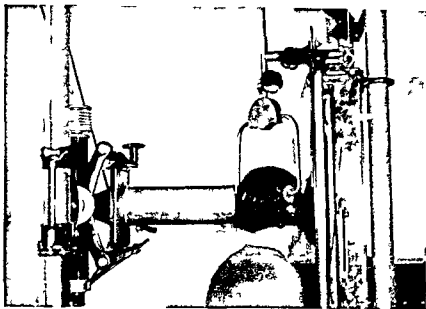


FIG 190 —Position for right oblique view

clears up the cause of the opacity. It also shows the depth of the frontal sinus.

In certain cases stereoscopic views in one or more positions are useful. Stereoscopic lateral views are of particular utility in locating wandering ethmoid cells.

These then are the six standard views used in this technique. They are ancillary to one another, and each forms a composite part of the whole. Moreover, though each view is primarily taken for a particular sinus or group of cells, they overlap the series giving at least two views from differing angles of each sinus. For instance, the occipito mental view is the primary film for the maxillary antra, but they are also shown from different angles in the vertico mental and lateral views. Again, the frontal sinuses are often very usefully visualised in the oblique views as well as in the occipito mental view.



FIG 191 —Right oblique view

It will also be seen that by using this technique a radiogram taken in any given one of these positions will be exactly comparable so far

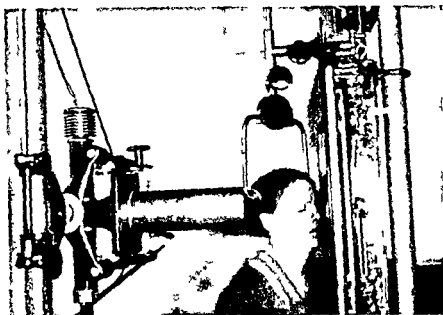


FIG. 192.—Position for left oblique view

as the normal shadows are concerned, with the corresponding radiogram of another patient. Thus, by this method, any variation from the normal will be at once apparent to the experienced radiologist. Necessarily, it takes a little time and experience to distinguish accurately each of the multiple

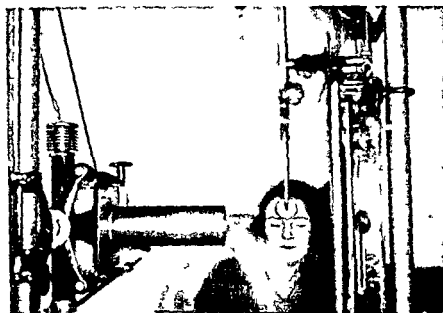


FIG. 193.—Position for lateral view

normal shadows in each view even when they are invariably projected into the same relative positions by the technique described above. One can imagine how unnecessarily difficult this becomes when they are thrown all over the film according to the prominence of the patient's nose, chin, or forehead and how next to impossible it is—under such conditions—to recognise pathological changes with any approach to accuracy.



FIG. 194.—Lateral view

It is entirely due to a haphazard technique that some otolaryngologists have until a few years ago placed little reliance on X-ray findings. Radiology properly employed can give more accurate information concerning the condition of the accessory sinuses than any other method of investigation, with the exception of radical operation and that exception only applies in the case of the anterior group of sinuses. The Central London Throat, Nose and Ear Hospital

treats a very large number of cases of sinus disease every year. Twelve years ago this hospital had no X-ray apparatus and for five years after its introduction only half a dozen cases per week were sent for X-ray examination. After the introduction of the upright stand technique the numbers steadily increased and to-day so great is the reliance placed on the X-ray findings by the laryngologist that the vast majority of all cases of suspected sinus disease at this hospital are referred for X-ray examination.



## CHAPTER IX

### DISEASES OF THE ACCESSORY NASAL SINUSES

#### SINUSITIS

SINUSITIS is one of the commonest diseases that flesh is heir to. The disease speaking broadly may be divided into two types hyperplastic and suppurative depending on the causative factor. At a later stage these two types may merge into one another depending on sequelæ of infection in the one case and on superadded infection in the other.

**Hyperplastic Sinusitis**—The commonest cause of this condition is a deviated septum. The constant obstruction to the free passage of air and mucus produces first of all hypertrophy followed by hyperplasia of the mucosa of the affected sinuses. It is a slowly progressive change usually commencing in the ethmoids.

The ostium becomes narrowed by the thickened mucosa and secretion may be retained and infected with a resulting superimposed acute suppuration. The periosteal layer of the mucosa may become so fibrosed as to constrict the blood vessels running in it with a resulting sclerosing osteitis of the bony walls, *this being most commonly seen in the ethmoid cells where the thin bony septa are sandwiched between two layers of mucosa*.

The thickening of the lining membrane may also result in the formation of polypi. Blocking of the ostium of the frontal sinus by thickened mucosa may result in the formation of a mucocele.

The X-ray appearances in a typical case are as follows. The nasal septum is usually seen to be deflected. The affected sinuses are opaque but the opacity is usually seen to be non homogeneous when a correct kilovoltage is used. The thickened mucosa is seen to have a sharply defined inner border the central portion of the sinus being comparatively transparent.

When multiple polypi are present they may so fill the cavity of the sinus overlapping each other's contours as to make the opacity homogeneous.

**Suppurative Sinusitis**—Suppurative sinusitis usually commences in the antrum. The commonest cause of this condition is coryza and there is usually an appreciable rise in the incidence of this condition in an influenza epidemic. Among the less common causes is the spread of infection from a septic tooth especially when the apex of the tooth bulges into the antrum and is only separated from it by the antral mucosa. Fractures of the antral wall with

effusion of blood into the sinus may also lead to suppurative sinusitis. The condition is common in hot dusty countries such as Queensland.

In the early stages of the disease the sinuses show a cloudy semi transparent ground glass appearance which is in marked contrast to the hard well defined edges of the fibrosed mucosa seen in hyperplastic sinusitis. At first the bony walls of the sinus are unaffected and show a normal definition. Exudate later collects in the sinus and the ostium may become blocked either through thickening of the mucosa round the ostium or through a plug of thick pus or mucus becoming impacted in it.

With this blocking of the ostium the antrum may become completely filled with fluid in which case of course no fluid level will be apparent and the sinus will show a homogeneous opacity on the radiogram. As the disease proceeds it spreads to the other sinuses and the hyperaemia causes a varying degree of osteoporosis of the bony walls with consequent blurring and lack of definition in the radiogram. In rare cases the infecting organism may penetrate the mucosa and invade the bone causing an osteomyelitis. This uncommon complication is most apt to occur in connection with the frontal sinuses.

If acute sinusitis yields to treatment in the early stage a radiogram taken later may show little or no variation from the normal but when the disease has lasted for some time some chronic thickening of the mucosa will result.

The early acute changes in the mucosa are sometimes difficult to distinguish from the appearances seen in allergic conditions but in allergic conditions usually all the sinuses are affected and the transient nature of the abnormal appearances together with the history will guide one.

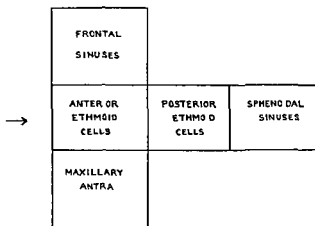


FIG. 195.—Diagrammatic representation of the sinuses viewed laterally and showing the superimposition of ethmoidal cells and sphenoidal sinuses as seen in the occipito frontal view in the plane of the arrow.

## RADIOLOGICAL INTERPRETATION OF SINUSITIS

The general method of interpretation will be most easily explained by taking a few typical cases afterwards dealing with special cases.

### SINUSITIS

**Gross Opacity**—It will be recalled that in Fig 185 showing the occipito frontal view two air spaces are seen on either side of

the perpendicular plate of the ethmoid. They consist of the superimposed shadows of the anterior and posterior ethmoidal cells and the sphenoidal sinuses from before backwards (Fig 195). Case I (Fig 196a) shows the space to the right to be opaque. Radiograms in the oblique position having demonstrated that the anterior and posterior ethmoidal cells are clear the vertico mental view (Fig 196b) shows the opacity of the right space to be due to an infected sphenoidal sinus. It will be seen therefore that starting with the occipito frontal view one has a double check on the condition of the ethmoidal labyrinth and the sphenoidal sinuses which works out something like a game of patience. Occasionally where there is unilateral deficient development of the sinuses their comparatively thick

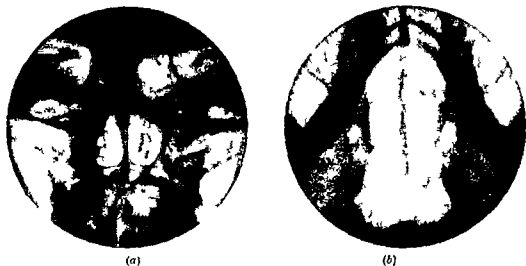


FIG 196 Two views of an infected right sphenoidal region. (a) shows an opaque ethmoidal region. (b) shows the opacity to be due to an infected right sphenoidal sinus.

bony walls will make the air space on that side opaque although no pathological lesion is present. The other views however will show the reason for this opacity also it must be borne in mind that if the angle is not exact the shadows of other structures will be thrown into the air spaces.

**Fluid levels**—Case II (Fig 197) illustrates another point. The horizontal line in the left antrum suggests that it is half full of fluid (Fig 197a). That is an assumption based on the horizontal upper margin. When the head is tilted to the left (Fig 197b) it is seen that the upper margin remains horizontal. Tilting to the opposite side (Fig 197c) has a similar result. Nothing but fluid will remain horizontal in an air-containing cavity when that cavity is tilted therefore the shadow is due to fluid. This is not an opinion or a vague assertion but a fact based on incontrovertible proof. Case III (Fig 198) shows fluid in a frontal sinus and in Case IV (Fig 199) fluid can be seen in the sphenoidal sinuses.



(a)



(b)



(c)

FIG 197—Demonstration of fluid level in the antrum by tilting the patient's head

At the stage of infection when cases are sent for X ray examination it is relatively uncommon to find a fluid level in the sinuses the incidence being about 6 per cent in the writer's experience (This figure does not include those cases who have a few hours after a proof puncture when residual fluid from the wash out is frequently seen) It is not frequency of the visualisation of a



FIG 198 Fluid level in frontal sinus

fluid level which matters but the determination of its presence or absence. Sometimes one sees a sinus which is homogeneously opaque with no fluid level apparent. In the majority of cases this is due to marked thickening of the mucosa or to multiple polyp but the radiologist cannot exclude in such a case the more remote possibility of the ostium being blocked and the sinus completely filled with fluid in which case of course no fluid level would be apparent. This possibility should be

mentioned in the radiologist's report since then the laryngologist knows that the sinus in question shows pathological changes from one of two causes and can determine by clinical examination which of the two conditions is present.

When a fluid level is visualised the radiologist cannot say what type of fluid it is i.e. whether it is mucus, mucopus or pus. Thick viscid pus flows slowly over into the horizontal position when the head is tilted and often has a convex upper margin but this is as far as one can go. Water casts as dense a shadow as pus.

**Thickened Lining Membrane**—A sinus may be translucent or opaque to X rays. Opacity may be due to a fluid content or to disease of the wall or to both and these excluding deficient development are the only three possibilities. The evaluation of normal translucency is a matter of experience based on the size of the sinus, its air content and the definition and clarity of the bony walls or foramina projected into the shadow of the sinus. With experience this can be accurately determined. In the case of an opaque sinus the extent to which proof can be given by radiology regarding a fluid content has already



FIG. 199.—Fluid in sphenoidal sinus.

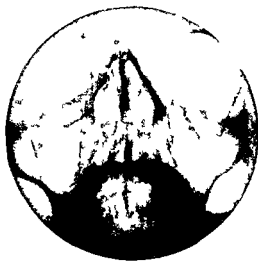


(a)



(b)

FIG. 200.—Chronic hyperplastic sinusitis in both antra with some fluid in the left proved by the tilted view (b).



(a)



(b)

FIG 201 Cyst of the left antrum

been shown. With regard to the third possibility, since two are proved, the third is simple. If a normally developed sinus with abnormal bony wall is opaque to X rays in all views and no fluid is present, the opacity must be due to thickening of the mucosa. But quite apart from this proof by elimination, not only thickening of the lining membrane, but the type of thickening is as accurately demonstrable as fluid. Case V (Fig 200a) shows marked chronic hyperplasia of the mucosa in both antra. It should be noted that whereas at the periphery the rays are traversing the entire thickness of the thickened mucosa, in the centre they are traversing the two layers of

mucosa on the posterior and anterior walls and consequently one sees a central comparative translucency. If the penetration be increased to the correct degree, there is present a thickening of the mucosa in both antra and at the same time a horizontal level in the left antrum. The skull is tilted (Fig 200b) and the thickened membrane tilts with it, but the horizontal line in the centre of the left antrum remains horizontal and therefore this sinus in addition to thickened lining membrane contains fluid.

Cyst. Case VI (Fig 201a) shows a different type of case. One sees



FIG 202.—Solitary polypus in left antrum



FIG. 203 — Polypus and chronic hyperplasia of mucosa in right antrum

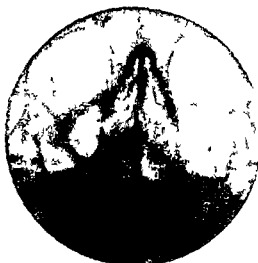


FIG. 204 — Acute infection of left antrum  
Compare with Fig. 203

an opacity occupying the lower half of the left antrum with a convex upper border but there is a small gap between the upper part of its outer border and the antral wall. On tilting (Fig. 201b) the upper margin tends to remain horizontal but does not quite succeed in doing so and the gap still persists. It is therefore fluid but encysted fluid and the obvious deduction is a cyst.

**Polypi** — A solitary polypus in the left antrum is seen in Case VII (Fig. 202)



FIG. 205 — Acute infection of right sphenoid



FIG. 206 — Chronic infection of right sphenoid  
Compare with Fig. 205



FIG 07 Osteoporosis of the walls of the ethmoid cells in ethmoiditis

Case X (Fig 205) shows a similar acute or subacute ground glass type of opacity in the right sphenoidal sinus while in Case XI (Fig 206) is seen the chronic fibrotic type of hyperplasia also in the sphenoidal sinus

**Bone Changes**—A very virulent or long standing infection of a sinus besides producing visible changes in the mucosa may produce changes in the bony walls

Again the hyperæmia produced by sinusitis may cause a partial osteoporosis of the bone giving a hazy and blurred appearance to the outlines of the sinus. This is most commonly and earliest seen in the ethmoid cells where the thin bony partitions dividing the cells are attacked from both sides as in Case XII (Fig 207)

Finally a long standing infection coupled with ischæmia produced by fibrotic changes in the periosteum and mucosa may produce a sclerosing osteitis of the sinus wall as in Case XIII (Fig 208)

### TUMOURS

**Osteoma**—These tumours present no difficulty in interpretation. They are very opaque to X rays, are clear cut in outline and are commonly found in the frontal sinuses and ethmoids (Case XIV Fig 209)

Case VIII (Fig 203) also shows a polypus in the right antrum. In this picture is also seen a dense hard line around the periphery of this sinus. This is due to chronic hyperplasia of the mucosa. In acute sinusitis one sees, instead of this dense hard line, a hazy

ground glass appearance—Case IX (Fig 204)—which varies in opacity according to the duration of infection. It is caused by œdema and swelling of the mucosa. A slight degree of this œdematous swelling is often seen following antral lavage. Case



FIG 08—Sclerosing osteitis of wall of left antrum



**Carcinoma**—In the early stages of malignant disease in the sinuses carcinomatous invasion of the mucosa is radiologically indistinguishable from inflammatory thickening. Usually it rapidly invades the nose and the posterior group of sinuses on the same side. A marked unilateral opacity involving the sinuses on one side of the nose with no corresponding evidence of abnormality on the contralateral side may make one suspicious of malignant disease, but it is not until the wall of the sinus has been invaded and the commencement of bone destruction has begun that one can definitely recognize malignant disease of the sinuses on the radiogram.



FIG. 209.—Osteoma of left frontal sinus

**Dental Cyst**—These cysts not infrequently force their way upwards through the floor of the antrum and may attain a large size in some cases nearly filling the sinus. In a radiogram they resemble a solitary polypus except that having a fluid content the plane of their upper margin alters as the head is tilted.

**Mucocele**—The ostium of an ethmoid cell or frontal sinus may become occluded; secretion accumulates and the sinus expands with gradual thinning of the walls.

## CHAPTER X

### THE USE OF IODISED OIL IN THE DIAGNOSIS OF NASAL SINUS DISEASE

*Forestier* and *Sicard* opened up a new field of radiographic technique by introducing the radio opaque substance lipiodol (a compound of iodine and poppy seed oil) for use in examination of certain of the body cavities and in the last few years it has been employed with a certain amount of success in the radiography of the nasal sinuses. For this purpose the lipiodol is usually diluted to half strength with olive oil or liquid paraffin to render it less dense.

The method has been more widely applied in America than in this country where its use has been subjected to a good deal of criticism. *Campbell Smyth* in America says that he has yet to see contrast media advocated for sinus radiography by a well known roentgenologist. *W. I. Mullin* states that if the time spent in preparing the patient and instilling lipiodol were occupied in studying a well taken film with a radiologist skilled in this field of work an equal amount of information would be obtained in most cases while *A. W. Proetz* who introduced the displacement method said that given good radiograms the necessity for the use of lipiodol would be reduced to a comparatively small number of special cases.

### TECHNIQUE

There are two methods of introducing the lipiodol.

**Method of Direct Filling** —In this method the oil is slightly warmed and introduced by an ordinary cannula and syringe through the ostium of the sinus or by puncture. Preliminary shrinkage of the mucosa especially if hypertrophied may be necessary. Radiological examination should follow as soon as possible after the injection. This method is seldom used but occasionally where for instance a septate antrum is suspected it may be of great use.

**Method of Indirect Filling** *Proetz* in America has evolved a method called the displacement method for introducing lipiodol and medicinal drugs into the posterior group of sinuses.

The method is based on the fact that if a liquid be introduced into a cavity so as to submerge entirely the mouth of another cavity opening into it and if suction be then applied to the first cavity and the other openings closed the air sucked out of the second cavity is replaced by the fluid covering its mouth when the suction is released.

The technique is as follows. The patient lies supine with his head projecting beyond the end of the couch and his head extended until the chin is on a level with the external auditory meatus. About 5 c.c. of lipiodol is introduced into both nostrils, covering the ostia of the posterior group of sinuses. One nostril



FIG. 10

Occipito mental and erect co mental views after introduction of lipiodol by displacement method



FIG. 11

is closed a suction syringe is applied to the other and the patient told to say "K" quickly and repeatedly so as to close the nasopharynx. This suction is applied about six or eight times to each nostril and the patient is then X-rayed in the erect posture the lateral vertico mental and occipito frontal views being the most useful (Figs. 210 and 211).

### THE VALUE OF THE DISPLACEMENT METHOD

In localising or identifying an aberrant cell the method may be of great use and if the lipiodol enters a cell it certainly proves that the ostium is patent and one must admit that with a well taken straight radiogram the radiologist though able to detect accurately pathological changes in the mucosa cannot say whether or not the ostium is patent. But unfortunately the converse is not true for if the lipiodol does not enter a sinus it proves nothing. The direction of the ostium in relation to the suction stream, its size, submersion and position in relation to the sinus, the size of the sinus, the degree of

suction the skill of the operator and the co operation of the patient—all are factors to be taken into account and which may make a great difference in the filling of the sinuses quite apart from any question of pathology. But even considering the non filling of a sinus in terms of pathology only non filling may be due equally to absence of air in the sinus (as a result of polyposis or fluid exudate) as to blockage of the ostium and blockage of the ostium cannot occur without pathological changes in the sinus radiographically visible by the ordinary method.

After all blockage of the ostium is not a separate entity but a possible sequel to a pathological condition in the sinus which is visible in a well taken ordinary radiogram but which may easily be concealed in the displacement method by the dense superimposed shadow of lipiodol in an adjoining cell.

Again a deflected septum an hypertrophied turbinate or any obstructive abnormality whether pathological or developmental will cause poor or complete absence of filling quite apart from any pathological changes in the sinuses themselves. Furthermore an agger nasi cell or a fronto ethmoidal cell will usually be situated above the level of the lipiodol and therefore will not fill. It is important to recognise the presence of such cells and though easily demonstrated by well taken radiograms by the normal method they will usually be concealed by the overlapping shadows of the lipiodol filled cells in the displacement method.

It is obvious from the above that there is a very wide opportunity for error in claiming that because a sinus does not fill with the lipiodol therefore the ostium is blocked. In fact if one were to take ten normal individuals who as far as can be ascertained have never had any sinus disease it is doubtful whether two would show lipiodol in *all* the sinuses after displacement.

Having considered the question of patency or occlusion of the ostium and the value of the evidence on these points provided by this method let us next consider what it can show us regarding the condition of the mucosa in the sinus. Proetz points out that thickened mucosa is shown as a dark band on the negative between the lipiodol and the wall of the sinus. This is true if and only if an unobstructed view can be obtained of the sinus concerned. But if as not infrequently occurs in the ethmoid cells other cells containing lipiodol are interposed between the X ray film and the cell concerned one can neither see this particular cell nor its thickened membrane.

Again the amount of lipiodol which can be introduced into a sinus by this method is usually very small only just covering the floor and therefore under favourable circumstances it is only the condition of the mucosa on the floor which can be demonstrated and obviously not the condition of the other walls of the sinus.

Furthermore a partial septum a tooth root bulging into the floor of an antrum or a misplaced ethmoid cell encroaching on the floor of a frontal or sphenoidal sinus may simulate filling defects in the lipiodol shadow.

Moreover lipiodol especially when it has been kept some time and some of the iodine dissolved in the olive oil may itself cause irritation and a temporary thickening of the mucosa.

Finally as regards evacuation of the lipiodol by the ciliary action of the mucosa it is claimed that if a sinus retains lipiodol after seventy two hours it is proof of pathology. The writer has frequently seen such retention in apparently normal individuals with no clinical history of sinusitis and on whom the displacement method has been carried out for purposes of experiment. It is particularly difficult for a radiologist to believe that any part of the body runs to a time table. The emptying time of the pulmonary alveoli after the introduction of lipiodol is such a very variable factor that one could not base a pathological inference on such evidence and it does not seem reasonable to attempt to do so in the sinuses.

We know that heat and cold and the presence of fluid have an effect on the activity or otherwise of the cilia the size and position of the ostia the effect of gravity and the amount of lipiodol introduced must also have an effect on the emptying time.

Though from the therapeutic standpoint the value of the method may be great from the radiodiagnostic point of view save in exceptional cases the method is more apt to conceal than reveal. Good radiograms well taken and well interpreted by an experienced radiologist are very much more accurate and reliable than radiograms taken by the displacement method.

Finally one very important point may have been lost sight of by otolaryngologists who practise this method and that is the danger of spreading infection. The ostium of the antrum is close to its upper extremity and it therefore becomes nearly its lowest point when the patient is in the exaggerated supine position. If the sinus contains pus the ostium will be covered by it during the time the displacement operation is taking place. Some of the pus therefore is very apt to be withdrawn by the suction and introduced into the posterior group of sinuses or into the Eustachian tube with the lipiodol. The method therefore presents dangers of spreading infection as well as of faulty diagnosis.

suction, the skill of the operator and the co operation of the patient—all are factors to be taken into account and which may make a great difference in the filling of the sinuses, quite apart from any question of pathology. But even considering the non filling of a sinus in terms of pathology only, non filling may be due equally to absence of air in the sinus (as a result of polyposis or fluid exudate) as to blockage of the ostium, and blockage of the ostium cannot occur without pathological changes in the sinus, radiographically visible by the ordinary method.

After all blockage of the ostium is not a separate entity, but a possible sequel to a pathological condition in the sinus, which is visible in a well taken ordinary radiogram but which may easily be concealed in the displacement method by the dense superimposed shadow of lipiodol in an adjoining cell.

Again, a deflected septum, an hypertrophied turbinate, or any obstructive abnormality, whether pathological or developmental will cause poor or complete absence of filling, quite apart from any pathological changes in the sinuses themselves. Furthermore, an agger nasi cell or a fronto ethmoidal cell will usually be situated above the level of the lipiodol, and therefore will not fill. It is important to recognise the presence of such cells, and though easily demonstrated by well taken radiograms by the normal method they will usually be concealed by the overlapping shadows of the lipiodol filled cells in the displacement method.

It is obvious from the above that there is a very wide opportunity for error in claiming that because a sinus does not fill with the lipiodol, therefore the ostium is blocked. In fact, if one were to take ten normal individuals, who as far as can be ascertained have never had any sinus disease it is doubtful whether two would show lipiodol in *all* the sinuses after displacement.

Having considered the question of patency or occlusion of the ostium and the value of the evidence on these points provided by this method let us next consider what it can show us regarding the condition of the mucosa in the sinus. Proetz points out that thickened mucosa is shown as a dark band on the negative between the lipiodol and the wall of the sinus. This is true if, and only if, an unobstructed view can be obtained of the sinus concerned. But if, as not infrequently occurs in the ethmoid cells, other cells containing lipiodol are interposed between the X ray film and the cell concerned, one can neither see this particular cell nor its thickened membrane.

Again the amount of lipiodol which can be introduced into a sinus by this method is usually very small, only just covering the floor, and therefore, under favourable circumstances it is only the condition of the mucosa on the floor which can be demonstrated, and obviously not the condition of the other walls of the sinus.

Furthermore, a partial septum, a tooth root bulging into the floor of an antrum, or a misplaced ethmoid cell encroaching on the floor of a frontal or sphenoidal sinus may simulate "filling defects" in the lipiodol shadow.

## CHAPTER XI

### ANATOMY OF THE TEMPORAL BONE

FOR RADIOLOGICAL purposes the temporal bones are conveniently divided into three parts, the squamous portion, the petrous bone, and the mastoid process

The squamous portion forms part of the floor and the lateral wall of the middle fossa. It is bounded above by the parietal bone, posteriorly by the mastoid portion of the bone, anteriorly by the greater wing of the sphenoid, and below by the petrous bone.

The outer surface is smooth and convex; the inner surface is concave and presents depressions for the convolutions of the temporal lobe of the brain and grooves for the branches of the middle meningeal artery. It is thin and scale like and casts, in the lateral view, a slightly less dense shadow on the radiogram than the other bones of the skull.

The zygomatic process projects from the lower part of the outer surface of the squamous bone. It has two roots, an anterior and a posterior. The anterior root terminates in the eminentia articularis; the posterior root is continuous with the temporal ridge.

The glenoid fossa is bounded in front by the eminentia articularis, and behind by the tympanic plate, which separates it from the external auditory meatus. It articulates with the mandible.

The petrous bone is pyramidal in shape and projects forwards and inwards at an angle of  $45^{\circ}$  with the sagittal plane of the skull, forming, with its fellow of the opposite side and the posterior border of the body of the sphenoid, the boundary between the posterior and middle fossæ of the skull. The bone, as its name denotes, is dense and hard, forming a strong protecting case to the complicated and delicate organs of hearing and balance contained therein.

The base of the petrous bone joins with the inner surface of the squamous portion and the mastoid process. Its apex is wedged in between the sphenoid and occipital bones. At the apex is the internal orifice of the carotid canal. The carotid canal passes outwards and backwards for a short distance and then bends vertically downwards to open on the inferior surface of the petrous bone. The petrous bone has three surfaces, an anterior, a posterior, and an inferior.

The anterior surface has an eminence near its centre which marks the position of the superior canal of the labyrinth. In front of, and a little to the outer side of this eminence is a depression consisting of a thin layer of bone

the tegmen tympani, which forms the roof of the tympanum and the mastoid antrum

On the posterior surface, near the centre, is the internal auditory meatus. It leads into a short canal about one third of an inch in length, which runs outwards and transmits the seventh and eighth cranial nerves

The inferior surface is rough and irregular, and forms part of the exterior of the base of the skull. On it appears the external orifice of the carotid canal



FIG. 212.—Normal mastoid and petrous portions of the temporal bone in the postero-anterior oblique view. The mastoid process is of the small-celled type. A Superior semicircular canal B Vestibule C Cochlea D External semicircular canal E Mastoid antrum F Internal auditory meatus

External to this is the jugular fossa, which varies in size and depth in different skulls and contains the bulb of the internal jugular vein and, with the occipital bone, forms the jugular foramen. At the outer limit of the inferior surface is the styloid process with the stylomastoid foramen at its base transmitting the seventh nerve and the stylomastoid artery.

The external auditory meatus extends from the tragus to the membrana tympani. It is about an inch in length. Its outer half is cartilaginous, its inner half osseous. The latter, which is the only part of radiographic importance, is directed inwards and slightly forwards. It tapers towards its inner end.



**The Middle Ear**—The middle ear or tympanic cavity is an irregular air filled space which communicates with the nasopharynx by the Eustachian tube. It contains a chain of thin movable bones the malleus incus and stapes which convey the sound vibrations transmitted from the membrana tympani on the outer wall of the cavity to the internal ear on the inner wall. The cavity consists of two parts the atrium or tympanic cavity proper and the attic above the level of the upper part of the membrane. Behind it communicates with the mastoid antrum and in front with the Eustachian tube.

The roof is formed by a thin plate of bone the tegmen tympani. The floor separates the cavity from the jugular fossa.

**The Internal Ear**—The internal ear or labyrinth consists of two parts the osseous and the membranous labyrinth the latter being contained within the former. The one with which we are solely concerned as radiologists is the former (Fig. 212).

THE VESTIBULE is the central part of the labyrinth and lies behind the cochlea and in front of the semicircular canals. On its outer wall is the fenestra ovalis closed by the annular ligament and the footpiece of the stapes. Its inner wall is perforated by several minute holes for the passage of the filaments of the auditory nerve.

THE BONY SEMICIRCULAR CANALS are situated above and behind the vestibule. Each describes part of a circle and they open into the vestibule by five orifices one of the orifices being common to two canals. The *superior canal* is vertical and is placed transversely to the long axis of the petrous bone. At one end it opens into the upper part of the vestibule. At the other end it joins the posterior canal to form the pars communis which opens into the inner part of the vestibule. The *posterior canal* also vertical is at right angles to the superior canal. At one end it opens into the lower part of the vestibule at the other it joins as described above the superior canal to form the pars communis. The *external canal* is horizontal and is situated at right angles to the other two. The two ends of this canal open into the upper and outer part of the vestibule.

THE COCHLEA resembles in shape the shell of a snail and consists of two and a half turns arranged in a spiral the bony axis of the spiral being termed the modiolus. The long basal turn has its commencement in the anterior wall of the vestibule. It forms a projection on the inner wall of the tympanic cavity known as the promontory. Close to the junction of the cochlea and vestibule are two openings the fenestra ovalis and the fenestra rotundum. The foot piece of the stapes fits into the former. The bony capsule of the labyrinth differs developmentally from the surrounding petrous bone. In the radiogram it is considerably more dense than the surrounding bone and shows a sharply defined outline.

**The Mastoid Process**—The pneumatization of the mastoid process is brought

about by the gradual extension—into the surrounding diploic bone of the process—of the mucosa of the mastoid antrum during the first five years of life. In the absence of disease during the period of development the extent of the cell formation in the two mastoid processes is symmetrical in 90 per cent of all cases, but the extent of pneumatisation in individual cases varies within very wide limits. Only the mastoid antrum may be excavated, the remainder of the process remaining diploic bone. The mastoid process in some instances may be of dense ivory hardness i.e. of the sclerotic type. This is a developmental, not a pathological condition. Again, infection of the mucosa during the pneumatisation period may cause a unilateral cessation of cell formation. This also produces a cessation of development in the size of this process.

In the typical cellular mastoid (Fig. 213) various groups of cells may be recognised: periantral cells around the mastoid antrum, sino-dural cells around the sino-dural angle, marginal cells external to and behind the sulcus sigmoideum, and terminal cells extending down into the apex of the process. In some instances cells also extend forwards over the temporo-mandibular joint and into the root of the zygoma. Cell formation may extend also upwards into the squamous portion of the temporal bone or inwards reaching in some cases the apex of the petrous bone.

It will be seen therefore that the location of the mastoid cells is by no means a constant factor, but a widely varying one, of which the radiologist must have a full cognisance. He must take radiograms in several positions so as to show any group of cells which may be present, and their relationship to surrounding structures.



FIG. 213.—Normal left mastoid process with cells extending into the zygoma (lateral oblique view)

## CHAPTER XII

### THE LABYRINTH AND PETROUS BONE

#### TECHNIQUE OF EXAMINATION

THE RADIOGRAPHY of the labyrinth, or internal ear, is a complicated process, because of the smallness of the part and the density and multiplicity of the overlying structures. The writer's original intention, in radiographing the labyrinth in 1927, was to endeavour to demonstrate the bony changes which take place in cases of otosclerosis. But the same technique has since proved extremely useful in showing fractures of the petrous bone, eighth nerve tumours, fistulae of the external canal, and other pathological conditions in this neighbourhood. The technique is based on that used by *Stemmers* in his original work on tumours on the auditory nerve. The apparatus used is the same as that which has been described for the radiography of the accessory nasal sinuses. As in that technique, the angles used must be exact and not "somewhere near" or "about."

Let it be presumed that a radiogram is required of the right labyrinth. The stool, with the patient sitting on it and facing the Potter Bucky, is displaced to the opposite, or left side. The head is then brought over towards the right until the head and neck are inclined at an angle of  $15^{\circ}$  to the long axis of the body. The head is then firmly grasped in its bitemporal diameter by the head clamp, and rotated on its vertical axis through  $45^{\circ}$ , so as to bring the petrous bone parallel to the film. If, however, a film were taken in this position, with the central ray horizontally behind the labyrinth, the shadows of the tympanum, temporo mandibular joint, and labyrinth would be superimposed, and to avoid this the central ray is tilted upwards through an angle of  $12^{\circ}$ . In this position the shadow of the foramen magnum and lower part of the occipital crest is thrown across the labyrinth and, to avoid this, the patient's head is so adjusted, that the orbito meatal line is inclined towards the feet at an angle of  $10^{\circ}$  with the horizontal. A point is then taken midway between the right external auditory meatus and the external occipital protuberance, and stereoscopic radiograms are taken on either side of this centre point. The positions are, of course, reversed for the radiography of the left labyrinth.

The radiograms, even in a normal labyrinth, are at first very difficult to read, but after a little practice one knows what to look for and can see every part of the canals and cochlea (*vide* Fig 212). The writer has found that the best way of gaining an accurate knowledge of the radiographic appearances of

the labyrinth is to pin firmly a piece of celluloid over the film on a viewing desk and to trace the labyrinth with a mapping pen. One will find that after studying a few dozen labyrinths in this manner the canals and cochlea can be seen standing out quite clearly from the surrounding structure.

### OTOSCLEROSIS

Seventy years ago *Toynbre* mentions "the establishment of the existence, as a disease, of membranous and osseous ankylosis of the stapes to the fenestra ovalis, one of the most common causes of deafness." During the years which have since elapsed a great deal of valuable microscopical research has been carried out upon the middle and inner ears of patients who have been the victims of otosclerosis, and much useful information has been acquired.

Many interesting clinical data have also been obtained and grouped so as to construct a fairly definite picture of the disease. Amongst these may be mentioned the hereditary factor; the onset of the disease, as a rule, in early life; the greater incidence in the female sex; tinnitus, nystagmus, vertigo, and increased bone conduction. The main symptom is gradually increasing deafness, which progresses by steps rather than by steady incline. Illness or pregnancy usually causes exacerbations. But, in spite of increased clinical knowledge of the condition, much requires still to be done, and it is here that radiology may be of some assistance. Practically nothing is known of the ætiology and treatment. Investigations of a long series of cases, extending over ten years, at the Central London Throat, Nose, and Ear Hospital, have established radiology as a useful ancillary method of diagnosis. Though it is improbable that the earliest changes around the oval window will ever be demonstrated radiographically (and therefore negative X-ray evidence does not imply the absence of disease), the extent and distribution of the disease can be demonstrated in the radiogram when the capsule is involved to any extent. The disease is almost always bilateral, but the extent of the bony changes, as in spondylitis, are frequently not proportional to the clinical signs and symptoms. The name itself is a misnomer, as both the microscopical and X-ray results show a porosis of the bones.

**Radiological Appearances.**—As previously explained, the bony capsule of a normal labyrinth shows a clear, sharply defined outline, standing out in marked contrast to the surrounding less dense petrous bone. In otosclerosis the first radiographic changes seen are rarefaction and consequent lack of definition of the basal turns of the cochlea. At a later stage all the turns of the cochlea may become affected. At the same time the bony capsule of the semicircular canals loses its sharp outline and becomes porous, swollen, and ill-defined, its margins tending to merge into the shadow of the surrounding petrous bone. In some cases there is a plaque of bone overlying the shadow of the modiolus. The author does not know the pathological significance of these abnormal

bony shadows in the cochlea but he has seen them in a few cases of otosclerosis and never in any other condition

On looking at a radiogram of very advanced otosclerosis one gathers at first the impression that the hazy blurred appearance of the labyrinth is due to faulty radiographic technique but on studying the detail of the surrounding petrous bone one sees that this is not so as the lack of definition is solely confined to the labyrinth and is due to changes in the capsule. In such a case there is usually in addition to the usual porosis of the basal cochlea well marked porosis of the capsule of the posterior labyrinth. The bone surrounding the superior canal may be less dense than normal decreasing gradually in density towards its periphery and gradually merging into the shadow of the surrounding petrous bone with no sharp line of definition between. The capsule of the cochlea also has a very blurred and hazy appearance. The author has seen these changes only in very advanced cases of otosclerosis.

Though the radiology of the petrous bone—so far as otosclerosis is concerned—is admittedly still in its experimental stages it is hoped that with continued research work and close co operation it will assist materially in

throwing light on one of the most abstruse difficult and disabling conditions with which the aurist has to deal

### TUMOURS OF THE PETROUS BONE

**Tumour of the Eighth Nerve**—This is a neuro fibroma the site of which is usually in the internal auditory meatus. When it attains a sufficient size it produces a smooth symmetrical erosion of the bone.

**Sarcoma**—These may produce erosion of the bone or if osteogenic new bone formation in addition to erosion. Fig 214 is of an osteosarcoma arising from the upper border of the petrous bone.



FIG 214—Sarcoma of the petrous bone

### FRACTURES OF THE PETROUS BONE

Basal fractures involving the petrous bones are sometimes not demonstrable in the ordinary projections. The technique described usually reveals them

## CHAPTER XIII

### THE MASTOID PROCESS

#### ACUTE MASTOIDITIS

**Etiology** —Acute mastoiditis is a sequel to infection in the middle ear. Acute suppuration of the middle ear is most common in children especially those who are poorly nourished and lack sufficient fresh air and sunlight.

The most common path of infection is from the nasopharynx through the Eustachian tube the commonest cause probably being infected adenoids. The immediate predisposing causes are most frequently the common cold, influenza and the specific fevers. Infection may also take place through infected water in public baths penetrating into the Eustachian tube. Less common causes are injuries to the membrana tympani either through the introduction of or unskilled extraction of foreign bodies. Infective conditions of the external ear such as furunculosis and fractures involving the mastoid process may also in occasional cases be the predisposing factor.

The continuity of the mucous membrane of the nasopharynx, Eustachian tube, middle ear and mastoid antrum facilitates the spread of infection the opposing physical forces being the ciliary action of the mucosa lining the Eustachian tube and the closure of the tube by congestion.

**Pathology** —In the early stages of a middle ear infection the Eustachian tube becomes blocked owing to congestion and swelling of its mucosa the air in the middle ear becomes absorbed and owing to negative pressure the tympanic membrane becomes retracted. At this stage the ear may return to normal with no infection of the mastoid process but if the disease progresses the mucous membrane of the tympanum becomes involved mucus is poured out and the drum commences to bulge outwards. In the third stage the mucous exudate becomes purulent and increases in quantity the tympanic membrane becomes inflamed and the bulge becomes more pronounced. The inflammation spreads to the attic and from there to the mastoid antrum. At this stage with a cellular mastoid and good radiograms the first X-ray evidence of mastoid involvement is the appearance of a hazy halo round the periphery of the antrum. Usually however this cannot be seen in a diploic or sclerotic type of mastoid.

Finally the drum ruptures or a paracentesis is performed and the pus escapes. Drainage through the rupture and down the Eustachian tube may lead to complete resolution but if the infection has been virulent the swollen mucosa in the attic and mastoid antrum may retain the pus and suppuration.

will then continue in the mastoid process. In the more virulent infections there may be necrosis of the bony walls of the middle ear and mastoid and the ossicles may be destroyed.

The occurrence of an acute mastoiditis as a sequel to otitis media depends to a large extent on the anatomical type of mastoid process present. The complicated network of cavities in a cellular mastoid is more prone to the spread of infection and very much more difficult to drain through a paracentesis opening and therefore an acute mastoiditis is much more likely to occur in such a mastoid than in one of the diploic or sclerotic type. But against this the cortex is thinner and the signs of acute inflammation appear earlier both clinically and radiographically in a cellular mastoid.

In an acellular mastoid, particularly one of the sclerotic type, the mastoid antrum has a thick layer of bone between it and the surface and only a thin layer, the tegmen antri, between it and the meninges. In such a mastoid both the clinical and radiological signs are slight and appear later than in a cellular mastoid.

Therefore an acute mastoiditis is more likely to occur but is much easier to detect in its early stages in a cellular mastoid than in an acellular mastoid but dangerous complications are more prone to develop in the acellular type.

### CHRONIC MASTOIDITIS

Chronic suppurative otitis media, which is the outward and visible sign of chronic mastoiditis, may be due to other conditions such as chronic sepsis in the nasopharynx, nose, or throat, in the tympanum or in the attic or in all these structures but, with the exception of the sinuses, the only cause with which we are concerned radiologically is the mastoid process.

In this condition there is continuous middle ear discharge for varying periods. At intervals there is a lessening or complete disappearance of the discharge accompanied by pain and tenderness over the mastoid and a rise of temperature. This is due either to exacerbation of the inflammation or blockage of the aditus. After a few days the discharge may recommence and the pain and temperature disappear.

As one would expect, X-ray examination shows the mastoid to be cellular in the majority of such cases.

### THE POST OPERATIVE MASTOID

In order that the radiologist shall understand the post operative appearances of the mastoid processes it is necessary for him to possess a knowledge of the main features of the cortical and radical mastoid operation.

**Cortical Mastoidectomy**—This is the operation of choice in cases of acute mastoiditis where there is persistent pyrexia, discharge, pain in the ear and

tenderness over the mastoid process. It is essentially an operation to drain the middle ear through the aditus without disturbing the middle ear. It is therefore used in those cases where the ossicles are unaffected and hearing is not permanently impaired. It consists in removing the cortex covering the lateral aspect of the process, opening all infected cells, and draining the mastoid antrum and the middle ear through the aditus ad antrum.

**Radical Mastoidectomy**—This operation is performed when there is well marked and permanent loss of hearing and when continuance of the discharge and other symptoms renders it necessary to eradicate the infected tissues or when there are symptoms of intracranial or labyrinthine complications.

Prior to the operation any septic focus in the nose or throat is removed. The operation consists essentially in throwing the middle ear, aditus and mastoid antrum into one smooth cavity, the remains of the ossicles, membrane and diseased mucosa being removed and the cavity drained through the external auditory meatus.

### SCOPE OF X RAY EXAMINATION OF THE MASTOID PROCESS

A brief consideration of the anatomical variations of and the pathology of the mastoid process indicates the scope of the information which is required of the radiologist by his surgical colleague. The required information may be summarised under two main headings as follows:

#### Anatomical Information

- (a) *Are the processes cellular or acellular?*
- (b) *If cellular, what is the distribution of the cells? Do they extend beyond the recognised normal limits? Is the cortex thick or thin? (Fig. 215)*
- (c) *If the processes are acellular, are they diploic or sclerotic?* This latter



(a)



(b)

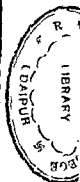


FIG. 215.—Normal mastoid processes seen in the postero-anterior or oblique view. (a) Large-celled type with their cortex and zygomatic cells. (b) Mixed-cell type with thick cortex.



type is said to be a developmental and not a pathological type as one might infer from its name

(d) *Are the processes symmetrical?* Developmental asymmetry is rare but asymmetry may be the result of disease in early life interfering with the extension of the antral mucosa into the surrounding bone and thereby preventing pneumatization of the process. Such a mastoid process may be recognised by the fact that besides being poorly pneumatized it will be smaller than its fellow of the opposite side

### Pathological Information

At first sight this subdivision would appear to be of much greater importance than the former but this is far from being the case. The type of mastoid present and the distribution of the cells are of paramount importance to the surgeon not only from the operative point of view but from the point of view from which he evaluates the patient's symptoms and the radiologist who wishes to give a useful opinion on the X ray findings cannot pay too much attention to the influence of anatomical type on the pathology and possible complications of mastoiditis. The only method short of operation by which the surgeon can tell whether a mastoid is pneumatized or not is by X rays and the difficulties of diagnosis both clinical and radiological are very much increased in a diploic or sclerotic mastoid.

A sharp distinction should be made between the cellular and acellular mastoid when considering the scope and accuracy of X ray diagnosis in mastoiditis. To use a nautical simile the radiogram of a cellular mastoid may be likened to a clear night at sea when the look out man in a ship's bows can give accurate and reliable information to the bridge.

On the other hand a diploic mastoid to the radiologist is like a heavy mist with three hundred yards' visibility and a sclerotic mastoid a thick fog when the navigating officer can scarcely see the stem head. There is however one important difference in this simile for whereas in thick weather at sea vessels proceed at reduced speed the surgeon when informed by the radiologist that he is dealing with an acellular mastoid goes full steam ahead. He does not wait for any further information from the radiologist and realising the much greater danger of grave complications operates at an earlier stage and on fewer signs and symptoms than he would in a cellular mastoid. The occurrence of an acute mastoiditis as a sequel to otitis media depends to a large extent on the anatomical type of mastoid present. Thus the complicated and communicating network of cavities in a *cellular mastoid* is more prone to the spread of infection and very much more difficult to drain through a paracentesis opening. Therefore an acute mastoiditis is much more likely to occur in such a mastoid than in one of the diploic or sclerotic type. But against this the cortex is thinner and the signs of acute inflammation appear earlier both clinically and radiographically in a cellular mastoid. In an *acellular*

*mastoid*, however, the mastoid antrum has a thick layer of bone between it and the surface, and only a very thin layer of bone, the tegmen antri, separates it from the meninges. Because of this, dangerous complications are very much more likely to occur, owing to the infection taking the path of least resistance. In the early stages of such a case, when the clinical signs are doubtful, the report of the radiologist that the mastoid is acellular will immediately reveal to the surgeon the reason for the doubtful signs, and is all he wants to know. In such a case, where, owing to the density of the overlying bone, the mastoid antrum may not be visible, the radiologist, when asked about evidence of infection, should point to the bony "fog" and say, "I do not know," and he might add with emphasis, "I do not want to know," because generally the first sign of infection he will see in such a process is bone destruction, and by that time the patient's life will be in imminent danger.

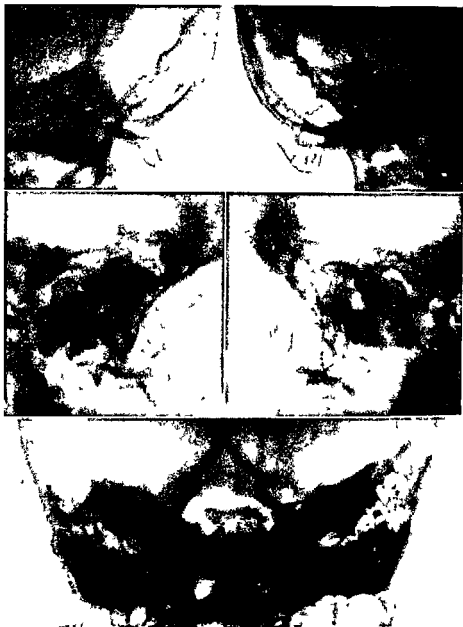
### TECHNIQUE OF EXAMINATION

The writer takes five radiograms altogether which give three separate and distinct views of the mastoid process, each of them a necessary one. It is convenient to use a vertical sinus stand, with Bucky grid, and, as in sinus radiography, the angles used must be exact and not "somewhere near," or "about." The five views are seen in Fig. 216.

(1 & 2) *The Postero-anterior Oblique Position*—The head is grasped in the clamp and rotated through  $45^{\circ}$ . This brings the petrous bone parallel to the film and throws the mastoid process clear of overlapping shadows. The tube is centred over the process. This view shows (a) whether the process is cellular or not and (b) the thickness of the cortex and the extent to which the cells spread inwards into the petrous bone.

(3 & 4) *The Lateral Oblique Position*—The head is in the lateral plane, and the tube is tilted downwards  $30^{\circ}$  and centred on the external meatus nearest the film. This view gives a  $90^{\circ}$  variation of the perspective angle from the previous one, and shows the distribution of the cells in the antero-posterior plane. It demonstrates the relationship of the cells to the knee of the lateral sinus, and whether they extend upwards into the squamous or forwards into the zygoma. It shows the thickness of the tegmen and whether any tegmen cells are present.

(5) *The  $30^{\circ}$  Fronto-occipital View*—This is sometimes a very useful view. It not only shows both petrous bones and any cell extension into them, but it gives what amounts to an axial, or end-on view of the two mastoid processes on the same film. The utility of this is that in the early stages of infection only a very slight hazy lack of translucency of the cells is seen in the other views and is very much increased in this view, owing to the fact that the projection through the whole length of the cells is much greater than a transverse section.



R

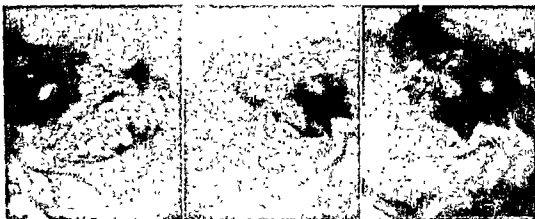
L

FIG. 216 —A case of early acute right mastoiditis indicating the value of the three views. In the postero-anterior oblique view only slight cloudiness is seen in the right mastoid (top pair). In the lateral oblique view (middle pair) the right mastoid air cells are also cloudy in appearance, but in the fronto-occipital view (bottom) this change is much more marked.

## RADIOLOGICAL INTERPRETATION

**Acute Mastoiditis in a Cellular Mastoid.**—In the great majority of cases of acute otitis media there is to be seen in the radiograms some lack of definition of the mastoid antrum, due to œdema of the mucosa (Fig. 216). If the condition progresses and the infection of the mastoid process spreads, the periantral cells and, later, the other cells of the process become hazy in outline and more opaque than their fellows of the opposite side, and finally the cell walls become invisible. In cases of bilateral infection or developmental asymmetry, the radiologist must rely on the lack of definition of the cell outlines and his experience of the normal translucence of the mastoid cells.

The distribution of the mastoid cells should be carefully noted and especially the relationship of any cells to the lateral sinus, the tegmen antri, or the zygomatic process.



(a)

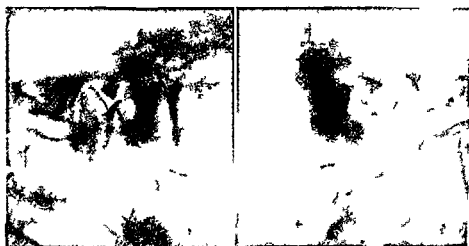
(b)

(c)

FIG. 217.—A case of left mastoiditis. (a) Normal right mastoid. (b) Acute mastoiditis in a cellular mastoid. Note blurring of the squamous cell. (c) The same, six months later, now showing a sclerosing osteitis.

Attention should be drawn to any cells mesial to the labyrinth or extending upwards into the squamous portion of the temporal bone. These cells, if infected, have to be carefully searched for. Infected squamous cells often show little difference in radiographic density from the surrounding bone, only a slight localised blurring of the bone structure being discernible. It is obviously of the greatest importance that the surgeon should know of the presence of such cells. The use of a powerful magnifying glass together with comparison with the cell distribution on the opposite side will usually reveal their presence. Infected cells bordering on and posterior to the lateral sinus should be noted, and in this connection stereoscopic views will often be found useful.

**Chronic Mastoiditis in a Cellular Mastoid**—Chronic suppurative otitis media with a constant or intermittent discharge from the middle ear is a common disease usually commencing in childhood. The name is misleading as it tends to focus attention on the middle ear with a consequent tendency to local treatment whereas the real site of the infection frequently lies at a distance from the middle ear. A persistent infection anywhere from the nasopharynx to the mastoid air cells may be responsible for a middle-ear discharge. Where the condition is the result of a chronic mastoid infection a radiogram of the affected process will show it to be opaque (Figs 217-18). The cell walls will be seen to be very blurred and indistinct or will have disappeared altogether. In such cases the phrase "breaking down of the cell



(a)

(b)

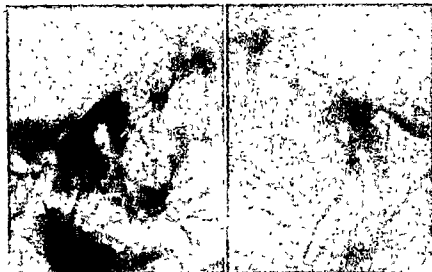
FIG. 218—(a) Normal right mastoid process. (b) Left mastoid process of the same patient showing a chronic sclerosing mastoiditis of ten years' duration.

walls" is frequently used somewhat loosely and inaccurately. On the one hand the cell walls may actually be eroded but on the other long continued infection and consequent hyperæmia will cause osteoporosis and therefore transparency of the cell walls and there are no radiological means of distinguishing between the two conditions. It is better therefore to describe this appearance by saying that the cell outlines are invisible (Figs 218 and 219).

**Mastoiditis in an Acellular Mastoid**—The difficulties of diagnosis are greatly increased from both the clinical and radiological points of view. In a diploic mastoid process some cloudiness of the mastoid antrum may be seen and there may also be present a few periantral cells which show changes. In a sclerotic mastoid however the mastoid antrum will probably be concealed by the dense overlying bone.

Speaking generally, in a case of acute mastoiditis in an acellular mastoid process, the radiologist has served his purpose in the case when he has informed the surgeon that the mastoid is acellular. At the same time he should be careful to make quite clear the fact that, because there is no radiographic evidence of infection of the process, this by no means indicates that disease is not present. In a sclerotic mastoid and frequently in a diploic mastoid no X-ray signs whatever are present until actual bone destruction commences, and by that time very serious complications may have arisen.

In such cases the clinical signs are often slight, but the risk of serious complications is very much greater than in a cellular mastoid.



(a)

(b)

FIG. 219.—A case of chronic left mastoiditis. (a) Normal right side. (b) The cell outlines in the left mastoid are completely invisible.

The radiologist's report, however, that the process is acellular will give the surgeon the necessary clue to the paucity of clinical signs, and he will operate on considerably less evidence than he would in the case of a cellular mastoid. Early operation is essential in such cases, as the mortality of all intracranial complications of acute mastoiditis is high.

The important radiological point in such a case, therefore, is that the mastoid process is acellular; and the absence of X-ray evidence of disease should be ignored, if it in any way conflicts with the clinical suspicions.

### COMPLICATIONS OF MASTOIDITIS

**Cholesteatoma.**—This is a chronic condition in which the middle ear, mastoid antrum, and mastoid process contain packed masses of flaky epithelial

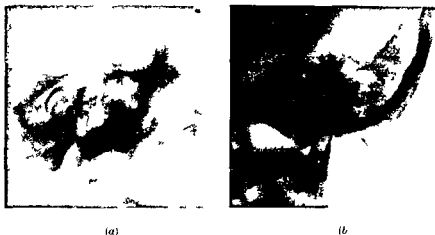


FIG 220—Cholesteatoma of the left mastoid (a) Lateral oblique view (b) Postero-anterior oblique view

debris which may grow to a large size. It is often accompanied by great destruction of bone, the dura mater may be exposed with the formation of an extradural abscess, or the inner tympanic wall may be eroded with exposure of the membranous labyrinth. The X ray appearances are those of a smooth walled relatively translucent cavity in the mastoid process, usually with a sharply defined margin (Figs 220 and 221).

**Intracranial Complications**—There is no direct X ray evidence of these complications. In some cases one may see infected tegmen cells and evidence of infection of the bone of the tegmen. Occasionally one may see actual perforation of the tegmen.



FIG 221—Two cases of cholesteatoma of the mastoid process (postero-anterior oblique view)

**Fistula** —When in chronic suppuration of the middle ear infection invades the internal ear the spread is usually through the apex of the external canal on the inner wall of the aditus. Occasionally this breach in the capsule of the external canal can be seen in the radiograms.

**Zygomatic Mastoiditis** —This condition as its name denotes may be met with where the mastoid is infected and pneumatization has extended forward into the root of the zygoma. In rare cases these infected cells may rupture into and destroy the temporo mandibular joint.

**Apical Petrositis** —Occasionally well marked cell formation may be seen in the petrous bone mesial to the labyrinth the chain of cells sometimes extending even to the tip. Where such cells become infected the X ray appearances are similar to those seen in the cells normally situated in the mastoid process. But in cases where the cells are small and few all that may be discernible in the radiogram is a very faint blurring of the bony structure or again there may be no visible X ray change whatever.

## THE POST-OPERATIVE MASTOID

When the discharge persists for longer than the usual time after a mastoidectomy and no cause can be found for this persistence in the nasopharynx or elsewhere an X ray examination may be required of the mastoid process. A very careful search should be made in such a case for infected unopened cells. The cells most easily missed at operation are those

situated in the squamous portion of the temporal bone in the tegmen antri along the posterior mental wall backwards over the lateral sinus and forwards in the root of the zygoma (Fig. 222). Stereoscopic views in all planes are often necessary in such cases and sequestra should be carefully looked for.



FIG. 222.—Post-operative mastoid. Petro-squamous view showing unopened cells (→).



*PART THREE*

THE BONES AND JOINTS AND SOFT TISSUES

BY

S L BAKER Ph D MSc, MRCS, LRCP, DPH  
I CAMPBELL GOLDING, MB, ChM, MRCP DMRE  
E DUFF GRAY MA, MD, FFR, DMRE  
H K GRAHAM HODGSON CIO, MB, BS, FRCP FFR DMRE  
DONALD HUNTER MD FRCP  
M H JUPE BA, MRCS LRCP, FFR DMRE  
PETER KERLEY, MD, FRCP, FFR DMRE  
R E ROBERTS BSc MD DPH, FFR, DMRE  
S COCHRANE SHANKS MD FRCP FFR  
CECIL G TEALL MB, ChB FFR  
T IANE TIERNEY, MRCS, LRCP, DMRE  
E ROHAN WILLIAMS MD FRCP, FFR DMRE

## PART THREE

### THE BONES AND JOINTS AND SOFT TISSUES

#### SECTION I

#### THE NORMAL BONES AND JOINTS OF THE EXTREMITIES

BY

D COCHRANE SHANKS, M.D., F.R.C.P. F.F.R.

#### CHAPTER XIV

#### NORMAL BONES AND JOINTS

#### GENERAL

A KNOWLEDGE of the normal radiographic appearances of the bones and joints is essential to accurate radiographic interpretation. Equally important is a knowledge of anatomical variations, and alterations in the radiographic appearance due to variation in the position in which the part is radiographed. Certain standard positions have been generally accepted as being the most suitable in which to demonstrate the various bones and joints, and these will be indicated in their appropriate sections. For routine work it is important that a set of standard positions be adopted. Laziness in this leads to doubt in interpretation, and in some cases, such as that of the elbow joint, to appearances so misleading as to provoke misdiagnosis.

There are certain further elementary rules in routine examination of bones and joints which require emphasis.

(1) At least two views of the part under examination should always be obtained if possible. These should be at right angles to each other. Further views are frequently necessary. Failure to observe this rule gives rise to serious risk of missing, say, a fissure or fracture without displacement.

(2) Stereoscopy forms a useful alternative in cases where two right angled views are not possible (e.g. hip joint), and frequently it is advisable to use stereoscopy in addition to views in two planes, or to take both views stereoscopically.

(3) The bone or joint on the other side should always be examined in addition to the one in question, for comparison.

(4) Fluoroscopy should play no part in the examination of bones or joints except in the case of setting or manipulating fractures and then only in very exceptional cases

### THE BONES AND JOINTS IN ADULTS

The structure of these should be clearly seen in a radiogram of good quality

In the Shaft the cortex the underlying cancellous bone the medullary canal and the nutrient foramen are visible The normal periosteum is not

The cortex presents a uniform opacity with a regular subperiosteal margin (except where muscular and tendinous insertions produce irregularity) and a less defined but easily visible internal limit where it blends with the underlying cancellous tissue The cortex is thickest in the middle zone of the shaft and diminishes evenly in thickness towards the ends of the shaft where it becomes continuous with the delicate cortex of the ends of the bone

The cancellous bone forms a comparatively thin layer in the shaft enclosing the medullary canal Its mesh like trabeculation can be seen through the cortical shadow

The medullary canal is often invisible and when seen shows only as a poorly defined zone of increased translucency running along the centre of the shaft

The nutrient foramen is not always seen but where visible it appears as a fine canal running through the cortex into the medulla As it runs inwards it is directed obliquely away from the growing end of the bone It may simulate a cortical crack

In the Ends of the Long Bones the cortex diminishes in thickness when followed from the shaft to an extremely thin shell of compact bone covering an expanded mass of cancellous bone It is thickest under the articular cartilage at that portion in other words where it receives a more localised and so increased stress from the bones with which it articulates This rather thicker subcartilaginous portion is noteworthy for its smooth regular contour The extra articular portions on the other hand tend to show mild irregularity of contour at the sites of ligamentous attachment In this portion of the bone its strength as a weight and stress bearing structure depends on its cancellous bone In place of the delicate irregular cancellous bone reticulum seen in the shaft there is present a specialised regular architecture so arranged as to receive and withstand the stresses to which it is subjected This structure particularly marked in the neck of the femur can be seen in some degree in the ends of any of the weight bearing long bones

The Joints —The articular cartilages are normally undifferentiated from the soft tissues generally in a radiogram as are the inter articular fibro cartilages where present The thickness of the former is indicated by the width of the joint space the space separating the ends of the subcartilaginous cortices in a

joint This joint space varies according to the size of the joint being wider in the larger joints Its width is fairly constant in the same joint in different individuals with increasing age the joint space tends to become narrower In erosion of the articular cartilages from disease is seen in a radiogram as a narrowing of the joint space

The normal synovial membrane is invisible in a radiogram Its outline becomes apparent in some joints when the joint cavity is distended with effusion, blood or pus The capsular ligament is likewise undetectable, with the exception of the ligamentum patellæ in the knee joint

**Epiphyseal Scar**—The site of the epiphyseal cartilaginous plate is frequently visible throughout life, in the form of the "epiphyseal scar" a thin dense transverse line across the bone at the junction of shaft and extremity In addition to this epiphyseal scar, one or more similar lines are sometimes seen at varying distances up the diaphysis These lines of arrested growth were first described in relation to rickets and have been held by many authorities to represent zones of bone deposition in the healing stage of rickets Multiple zones were accounted for by remissions in activity Numerous workers noted that intermittent administration of phosphorus lead, arsenic and other poisons produced these lines *Harris* has questioned the accuracy of the "line test" as a sign of healing or healed rickets and has shown that such lines develop after any acute illness particularly pulmonary affections A full bibliography is given in his paper He accounts for the phosphorus and arsenic lines as being the result of their toxic effects The subject is discussed further under the section on Lead Poisoning

**Compact Islands**—These are small round or oval dense nodules of bone which are occasionally seen in the cancellous tissue They are usually 2-4 mm in diameter, situated at the intersection of large trabeculae and are most common in the wrist and foot Their cause is unknown and their significance nil They may be multiple and then constitute the condition known as osteopoikilia condensans disseminata

## THE BONES AND JOINTS IN CHILDREN

In children, the striking feature in the long bones is the differentiation into diaphysis and epiphysis

The Diaphysis or shaft shows in very young children a relative thinness of the cortex In older children this disproportion diminishes gradually until the adult ratio is reached In most of the long bones the metaphysis is remarkably broad relatively to the middle of the diaphysis, particularly at the growing end The end of the metaphysis is normally convex, and in the early stages roughly nodular in contour The epiphyseal plate cannot be seen

The Ossific Nucleus of the Epiphysis appears first as one or more tiny granular opacities These rapidly fuse and enlarge into a round or oval mass of

cancellous bone which normally presents an irregular nodular contour similar to that seen in the end of the metaphysis. As ossification develops further this nodular contour disappears and the epiphysis assumes adult contours. A recognition of these normal irregularities is of importance to distinguish them from pathological lesions.

As the epiphyses forming the joint are to a degree varying with the age cartilaginous the joint space seen in the radiogram is wide. Its width is greatest in infants and diminishes progressively with epiphyseal ossification. This wide spacing of the visible bony landmarks makes it a matter of some difficulty at times to be certain of the presence of dislocation and epiphyseal separations and in this respect radiograms taken in standard positions are essential. The most pronounced instance of this is in the elbow joint where an oblique lateral view gives an appearance closely simulating a separation of the capitellar epiphysis of the humerus.

The Joint Capsule cannot be differentiated from the underlying cartilaginous epiphysis but is usually seen differentiated from the overlying muscles and fatty subcutaneous tissues in children of 2 or 3 years.

The Accessory Epiphyses frequently begin to ossify by multiple ossific centres. Normally these fuse to form a plaque of bone whose deep surface in many epiphyses becomes markedly notched or cogged this surface engaging reciprocally with a similar notched surface on the metaphysis. The space between represents the active ossifying cartilage.

**Times of Ossification of the Long Bones**—There is great difference of opinion between different authorities about the times of appearance and fusion of the epiphyses of the long bones.

In the succeeding pages the figures given represent an average struck between the data given by the following authors: *Gray, Morris, Scudder, Poland, Cohen, Cunningham, Frazer, Paterson, Spalteholz, Piersol, Quain, Buchanan, Roberts and Kelly and Ashurst*.

*Paterson* in his radiological survey found that ossification was as a rule earlier in females (up to six months in appearance and five years in fusion of ossific nucleus). He also found that the dates of fusion in his series were one to three years earlier than the generally accepted times.

## CHAPTER XV

### NORMAL BONES AND JOINTS OF THE UPPER EXTREMITY

#### THE HAND AND WRIST

**Standard Positions.**—The two principal standard positions for the hand and wrist are the dorso palmar and lateral. The former view may be taken with the hand in radial or ulnar deviation, or in the midway position. To obtain a true lateral view of the thumb, the hypothenar eminence should be raised from the film by a pad. A palmo dorsal view may be obtained by pronating the forearm fully and placing the dorsum of the thumb on the film.

#### THE PHALANGES

**Ungual Phalanges.**—The tip of the ungual phalanges appears in a dorso palmar view as an expanded heart-shaped extremity with a rough margin. The lateral view shows this to be on the palmar aspect of the phalanges, and it shows considerable variations in its outline. The shaft tapers from base to tip.

The basal articular surface shows a double concavity separated by a slight dorso-palmar median ridge.

**Mid-phalanges.**—The head is bicondylar with a median dorso palmar groove. The articular surface of the condyles extends farther on the palmar surface than the dorsal. The shaft broadens as it merges with the base, and shows a gentle concavity on its palmar surface. In the dorso-palmar view several tiny nodules are at times seen laterally. These have been wrongly described as indicative of gout and rheumatoid arthritis. They represent the roughened line of attachment of the digital tendon sheath and are present chiefly in subjects of strong muscular and bony development.

The articular surface of the base is biconcave, separated by a slight median ridge.

**Proximal Phalanges.**—These show an appearance similar to that of the mid-phalanges, except that they are larger, their bases less regular, and their basal articular surfaces concave instead of biconcave.

**Phalanges of the Thumb.**—These present an appearance similar to the terminal and proximal phalanges of the fingers, except that they are thicker and more heavily formed.

#### THE METACARPUS

The Head of each Metacarpal Bone is rounded, the articular surface covering it being carried forwards onto two palmar condylar extensions. The neck and

shaft form a palmar concavity which is overhung by the condyles of the head. This relationship is of importance in the treatment of fractures of the metacarpal shaft.

The 5th metacarpal shaft has a palmo radial concavity.

The Bases of the Metacarpals have individual characteristics when viewed in a dorso palmar radiogram.

That of the 1st is saddle shaped articulating with the similar surface of the trapezium. It is often seen to overhang the trapezium in such a way as to simulate a subluxation.

The base of the 2nd is large and presents a prominent ridge on its ulnar side. It articulates with the trapezium, trapezoid, os magnum and 3rd metacarpal.

The feature of the base of the 3rd metacarpal anatomically is its spine. In a dorso palmar radiogram its shadow overlaps the ridge of the 2nd while in the lateral view it may be seen on the dorsal surface articulating with the os magnum.

The base of the 4th is small and rounded and articulates with the unciform bone and the 3rd and 5th metacarpals.

The base of the 5th bears facets for articulation with the unciform and 4th metacarpal and frequently projects beyond the unciform in a manner similar to the 1st metacarpal.

## THE CARPUS

The Trapezium and Trapezoid (os multangulum majus and minus) are irregularly cuboidal and their shadows overlap in an antero posterior view a feature which may simulate a fissure. A semi lateral view of the hand with the dorsum of the thumb on the film shows the former to best advantage.

The Os Magnum (os capitatum) is best seen in the dorso palmar view. It articulates with seven bones the 2nd, 3rd and 4th metacarpals, trapezoid, scaphoid, semilunar and unciform. Occasionally there is present on its lateral (radial) surface a small concavity which marks the site of the os centrale.

The Unciform (os hamatum) is triangular in shape the base towards the metacarpus. The hamular process which projects in a palmar direction is seen in the dorso palmar view as a prominent oval ring. The unciform articulates with the 4th and 5th metacarpals, the os magnum and the cuneiform.

The Scaphoid (os naviculare)—In the standard dorso palmar view the scaphoid is seen foreshortened owing to the obliquity of its long axis. This obliquity is increased on radial deviation of the hand and diminished by ulnar deviation. The latter position is therefore that of choice to demonstrate this bone. In the lateral view its outline is well seen and it then frequently presents a slight waisted appearance being narrower at its middle. This view shows the forward projecting tubercle clearly.

Occasionally there is to be seen in a dorso palmar view a small depression or pit on the ulnar side. This together with a similar depression in the adjacent surface of the os magnum marks the site of the os centrale.

**DOUBLE SCAPHOID**—This is held by some to occur but probably the appearance is always the result of an ununited fracture. The view that trauma is the cause of such cases is upheld by the fact that two well formed ossific nuclei have not yet been recorded. Even in those cases where ossification of the scaphoid begins in two discrete granules they invariably fuse to form one ossific nucleus.

The scaphoid articulates with five bones the radius semilunar os magnum trapezium and trapezoid.

Congenital absence of the scaphoid and semilunar has been described.

**The Semilunar (os lunatum)**—In the dorso palmar view the semilunar has an irregularly quadrilateral appearance and it is not until it is viewed laterally that its characteristic lunate outline is visible. Its proximal surface is convex in both views and its distal is concave.

The semilunar articulates with five bones the radius scaphoid os magnum the ulnariform (almost a linear facet) and the cuneiform. It does not articulate with the ulna but with the triangular fibro cartilage which separates it from the lower end of that bone.

Congenital fusion of the semilunar and cuneiform is said to occur as a rare abnormality in negroes.

**Cuneiform (os triquetrum)**—This bone is seen best in the dorso palmar view. Its shape is irregularly cuneate the narrow end of the wedge being directed distalwards. It is sometimes seen double but this is probably as in the case of the scaphoid always the result of an ununited fracture.

The cuneiform articulates with three bones the semilunar ulnariform and pisiform and with the triangular fibro cartilage which separates it from the lower end of the ulna.

**Pisiform**—The pisiform resembles in shape a large pea with a flat articular facet for articulation with the cuneiform. Its shadow in the dorso palmar view is usually superimposed more or less completely on that of the cuneiform. In the lateral its outline may be seen clear of any other bone. It has been described as double but in such cases the proximal bony nodule represents the pisiform secundarius (*vide infra*).

**Sesamoid Bones in the Hand**—Some of these are relatively constant and others of a varying rarity. The following figures compiled by *Pfitner* indicate the frequency of incidence of the common sesamoids while those of rare occurrence appear in the lower part of the table. The number present at each site is indicated in brackets.



## COMMON SESAMOIDS

1st metacarpo phalangeal joint (two)

5th metacarpo phalangeal joint (one or two)

I P joint of thumb (one or two)

2nd metacarpo phalangeal joint (one)

## RARE SESAMOIDS

3rd and 4th metacarpo phalangeal joints (one)

Distal I P joint of index finger (one)

Males

Constantly

78.4 per cent

72.9 per cent

49.2 per cent

Females

Constantly

76.5 per cent

72.9 per cent

45.9 per cent

} Less than 1 per cent

## THE LOWER ENDS OF THE RADIUS AND ULNA

It is convenient to consider these here, as they form an integral part of the wrist joint.

The Lower End of the Radius, seen best in the dorso palmar view presents a concave inferior articular surface which on the lateral side extends down to the tip of the styloid process. This surface is divided into two portions by a slight antero posterior ridge, forming facets for the scaphoid and semilunar respectively. Along the medial margin of this surface is attached the base of the triangular fibro cartilage, the apex of which is attached to the tip of the ulnar styloid process. Immediately above this margin on the medial surface of the lower extremity of the bone is another concave articular facet, the sigmoid cavity, for articulation with the ulna. Because of this concavity, a clear joint space is seldom if ever seen between these surfaces. On the outer surface of the lower end of the radius, at the base of the styloid process, a slightly raised irregularity is normally visible in muscular subjects. This marks the attachment of the tendon of the brachio radialis tendon.

The epiphyseal scar is frequently visible as a curved line of bone condensation the concavity towards the elbow.

In the lateral view the lower end of the radius is seen superimposed on that of the ulna. The view is of importance in the detection of Colles's fracture, and particularly in the estimation of the degree of reduction of that fracture after manipulation. The important normal landmarks in this respect are the gentle forward curve of the long axis of the bone at the lower end and the slight anterior inclination of the concave lower articular surface.

The Lower End of the Ulna.—In the dorso palmar view the prominent feature is the styloid process projecting downwards from the postero medial aspect of the lower extremity of the ulna. The tip of the styloid process forms the apical attachment of the triangular fibro-cartilage, and a line joining this to the medial border of the lower end of the radius indicates the plane of that cartilage. This line bisects a triangular space between the ulnar head and the carpus. The size of this triangular space varies with the position of the hand being increased by radial deviation of the hand, and *vice versa* and when opened out by radial deviation must not be mistaken for a subluxation or a tear of the internal lateral ligament of the wrist joint.

The distal half of the ulnar styloid is frequently seen as a separate rounded nodule. This has been ascribed to non fusion of a separate ossific centre for

the process, but in all probability this is always the result of an ununited fracture (*vide os triangulare infra*)

The distal surface of the ulnar head is normally at a slightly higher level than that of the lower end of the radius 1-3 mm as a rule

### ACCESSORY OSSICLES IN THE WRIST

As many as twenty four supernumerary carpal ossicles have been described. Many of these are doubtless merely spicules of bone detached from the carpus and metacarpal bases by trauma, and which have remained ununited. The following have some claim to morphological entity. They are arranged in their supposed order of frequency of occurrence (Fig 223)

(1) *Os Trapezoides Secundarium* — This the commonest accessory ossicle, is situated on the dorsal aspect of the carpus between the trapezoid and the base of the 2nd metacarpal

(2) *Os Centrale* — The os centrale occupies a site between the os magnum and the distal pole of the scaphoid. It is very rare and occurs according to *Pfitzner* in 0.5 of subjects. In the author's experience it is much rarer than this. The oval bed of the primitive cartilaginous os centrale frequently persists, however, and appears in a radiogram as a small oval translucency formed by the depressions on the adjacent os magnum and scaphoid. No trace

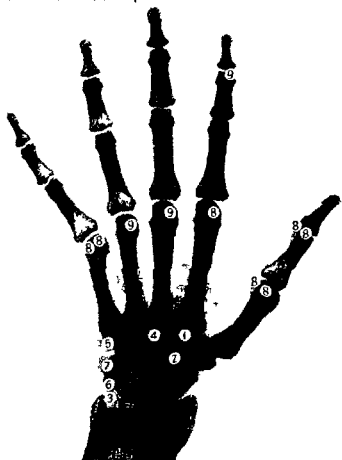


FIG 223.—Normal adult hand dorso palmar view, indicating the sites of sesamoids and accessory ossicles. (1) *Os trapezoides secundarium* (2) *Os centrale* (3) *Os triangulare* (4) *Os styloideum* (5) *Os vesalianum* (6) *Pisiform secundarium* (7) *Os ulnare externum* (8) Common sesamoids (9) Rare sesamoids

of the cartilaginous nodule normally persists. It is said to fuse with the lower pole of the scaphoid at an early stage in the development of the fœtus. Its homologue in the foot is the scaphoid bone.

(3) **Os Triangulare**—This is nearly always a post traumatic separation of the tip of the ulnar styloid process. This is a typical element in a Colles' fracture. It can occur with little or no damage to the lower end of the radius and the absence of evidence of old damage to the latter is no criterion that the so called os triangulare is not due to injury. However, as two bilateral cases have been recorded in which trauma has been said to have been excluded the existence of this ossicle as a morphological variation is a possibility.

The remaining ossicles are extremely rare.

(4) **Os Styloideum**—This appears as a separate ossicle at the tip of the styloid process of the 3rd metacarpal and lies on the dorsal aspect of the carpus.

(5) **Os Vesalianum**—This occupies a position close to the base of the 5th metacarpal.

(6) **Pisiform Secundarium**—This lies in the tendon of the flexor carpi ulnaris immediately proximal to the pisiform itself. An old transverse fracture will produce exactly the same appearance if as is usual it fails to unite.

(7) **Os Ulnare Externum**—This is described as being situated on the dorso medial aspect of the cuneiform bone.

## JOINT CAVITIES IN THE REGION OF THE WRIST

These are six in number and are of importance in the spread of a tuberculous or septic arthritis.

(1) **Inferior Radio-ulnar**—This lies between the lower end of the ulna and the triangular fibro cartilage and extends upwards between the lower ends of the radius and ulna.

(2) **Radio-carpal**—This joint space separates the lower end of the radius and the triangular fibro cartilage above from the proximal row of carpal bones (not including the pisiform) below. In the antero posterior view it is convex upwards.

(3) **The Basis cuneiform joint** space separates these two bones.

(4) **The Mid-carpal Joint** is a complicated cavity separating the upper from the lower row of carpal bones. In the dorso palmar view it is roughly S shaped and it sends prolongations upwards and downwards between the individual bones as far as their interosseous ligaments.

(5) **The Carpo-metacarpal Joint** separates the four bones of the distal carpal row from the bases of the four inner metacarpals, and as in the mid carpal joint, sends upward and downward prolongations between the individual bones.

(6) **Joint between the Trapezium and 1st Metacarpal**

### MOVEMENTS OF THE WRIST JOINT

Two joints only are responsible for the movements of the wrist joint, the radio carpal and the mid carpal. In flexion and extension both joints take part, the mid carpal having the larger share. Radial and ulnar flexion take place almost entirely in the radio carpal joint. A very slight degree of rotation is permitted in the mid carpal joint the head of the os magnum acting as a pivot. The parts played by these two joints in the movements of the wrist are evident in a radiogram, particularly those of ulnar and radial deviation.

With the Hand in the Normal Position the scaphoid and outer half of the semilunar are in contact with the radius, the remaining half of the semilunar and the cuneiform abutting against the triangular cartilage. The long axis of the unciform is directed obliquely.

In Full Radial Flexion the proximal row of carpal bones swing to the ulnar side, the scaphoid being foreshortened, the semilunar overlapping the radius to a greater extent, and the translucent triangle seen in the radiogram between ulna and cuneiform being widened. This increase in width must not be mistaken for a subluxation or torn internal lateral ligament. The long axis of the unciform becomes vertical, and that of the os magnum oblique.

In Full Ulnar Flexion the reverse changes take place. Thus the scaphoid casts a more elongated shadow and only its upper pole is in contact with the radius. The semilunar comes into full apposition with the radius and the ulnar cuneiform angle is closed. The normal obliquity of the unciform is increased.

### OSSIFICATION OF THE HAND AND WRIST

**Phalanges and Metacarpus**—The diaphyseal centres of ossification for these bones appear in the following order

Terminal phalanges	9th week of intra uterine life
Metacarpus	10th week of intra uterine life
Proximal phalanx	11th week of intra uterine life
Mid phalanges	12th week of intra uterine life
Mid phalanx of 5th digit	14th week of intra uterine life

*The epiphyseal centres appear and unite as follows*

	Appear	Unite
Metacarpal heads	2½–2¾ years	20th year
Bases of proximal phalanges	2½–2¾ years	18th 20th year
Base of ungual phalanx of thumb	2½ 2¾ years	18th 20th year
Bases of mid and ungual phalanges	3rd year	18th 20th year

Note that the 1st metacarpal resembles a phalanx in that it has a basal epiphysis and none for the head. This appears and unites at the same time as the heads of the other four metacarpals.

**Abnormal Metacarpal Epiphyses**—The base of the 2nd metacarpal is sometimes formed as a separate epiphysis. And in some cases where separated basal ossification is not achieved, evidence of an attempt at it is to be seen in

the form of an oblique notch on either side of the bone at the junction of shaft and base

The head of the 1st metacarpal sometimes forms as a separate epiphysis. When so the centre appears at the same time as those for the other metacarpals.

A less common epiphyseal centre is that at the base of the 5th metacarpal whilst they are very rare in the cases of the 3rd and 4th. They tend particularly to appear in cases of retarded growth such as Mongolian idiots. Their presence is normal in aquatic mammals and give in them increased mobility to the fore limb.

**Ossification of the Carpus** —At birth all the carpal bones are cartilaginous. Ossific nuclei appear in them at the following times after birth.

	A	Extremes
Os magnum	4th month	1st 6th month
Unciform	4th month	1st-6th month
Cuneiform	3rd year	2nd-4th year
Semilunar	4th 5th year	3rd-6th year
Trapezium	6th year	6th 7th year
Trapezoid	6th year	6th 7th year
Scaphoid	6th year	6th 7th year
Pisiform	11th year	9th 10th year

**Ossification of the Sesamoid Bones** —Those of the 1st metacarpophalangeal joint appear about the 14th year the others when present appear at a varying time later.

**Ossification of the Lower Ends of the Radius and Ulna** —These ossify as follows.

	Appears	Fuses
Radius	1st 2nd year	20th year
Ulna	5th 8th year	20th year

## THE RADIAL AND ULNAR SHAFTS

Both of these shafts show slight sinuous curvature and an appreciation of this is necessary for the correct estimation of displacement after a fracture. They should be viewed in at least two of three standard positions.

(1) **Palmo dorsal View** —The whole arm flat on the X ray couch anterior surface upwards the forearm in full supination (Fig 224). The ulnar shaft is concave medially in its upper half and the reverse in its lower. The whole shaft tapers from the upper extremity to the lower. The interosseous ridge is visible only in the lower half. The radial shaft shows at its upper end the prominent bicipital tubercle directed inwards and from this it describes a gentle outward convexity to the lower extremity. On the medial aspect of the shaft the interosseous ridge is a prominent feature. It must not be mistaken for cortical or subperiosteal thickening.

The radial and ulnar shafts overlap in this view only in their upper fourths.

(2) **Lateral Supinated View** —The whole arm on the X ray couch the humerus horizontal the elbow flexed to a right angle and the forearm in full supination (Fig 224). In this view the ulnar shaft presents a gentle curve the

convexity anteriorly. The radial shaft is slightly convex forwards in its upper third in the region of the bicipital tubercle. In the lower two thirds this changes into a slight concavity. The bicipital tubercle is barely visible on the contour of the bone and the interosseous ridge not at all. That of the ulna is, however, usually visible to some extent. The shafts are seen separate in their upper halves but overlap to an increasing extent as the lower ends are approached.

(3) **Lateral View**—The position is as in No. 2 except that the forearm is midway between supination and pronation with the palm downwards. In this view the two bones are seen separated to their maximum, their shafts converge one to another. Both interosseous ridges are clearly seen and the bicipital tubercle is seen in almost full profile.

### THE ELBOW JOINT

The three standard views for the elbow joint are similar to those for the forearm save that the central ray must be accurately centred over the joint. Particularly is this the case in the two lateral views when a slight variation in the centring or a very slight tilt of the elbow itself will give a very distorted appearance. The commonest fault lies in failing to have the upper arm resting horizontally on the couch in its whole length. This results in the shadow of the capitellum being thrown downwards and that of the internal epicondyle backwards.

**The Upper End of the Radius**—The neck of the radius forms a smooth

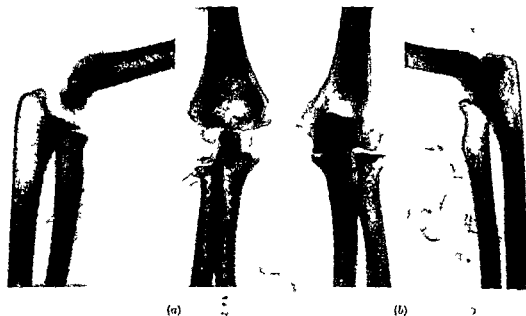


FIG. 24.—Elbow joint: (a) anteroposterior and medial views; (b) Child aged 5.

round cylinder, commencing below at the bicipital tubercle, and widening fairly abruptly into the head. The head in the form of a thick disc, has a concave upper articular surface for articulation with the capitellum of the humerus. This surface is continuous with the articular surface of the thick edge of the disc. The latter also has a cartilaginous covering, for articulation with the lesser sigmoid cavity of the ulna and the orbicular ligament. The articular margin descends farthest down on the inner aspect of the head. The outer portion of the radial head is seen, in an antero posterior view, to project laterally to some extent beyond the capitellum. This portion is normally relatively translucent and more rounded than the inner aspect. In all three views the shadows of the radial and ulnar heads overlap to some extent.

**The Upper End of the Ulna** —The structures of note in this are the coronoid and olecranon processes and the greater sigmoid cavity. All three are best studied in a true lateral view, but the actual width of the joint space is well seen in the antero posterior view.

The coronoid process ends in a rounded point. An actual spike is as a rule pathological, either the end result of a sprain of the tendon of the brachialis anticus or from osteoarthritis.

The greater sigmoid cavity forms in the lateral view a regular arc of about one third of a circle. In the antero posterior view it is saddle shaped, with a central ridge which rides in the trochlear sulcus of the humerus.

The olecranon process has a blunter tip than the coronoid. From this tip the upper surface of the olecranon passes backwards to join the posterior surface at an obtuse angle, usually a little more than a right angle. The superior and posterior surfaces of the olecranon have a considerable normal variation, depending on the development of the epiphysis. The edge dividing them is often slightly raised, forming as it does the main attachment of the triceps tendon. Frequently also a spur develops there, in that tendon. Such a spur may become long, be fractured, and subsequently form a separate ossicle in the tendon. An olecranon spur is directed obliquely forward, following the line of traction of the tendon.

**The Lower End of the Humerus** —In the antero posterior view the lower articular surface is seen to be sinuous in outline. On the inner half is the trochlea, with its central groove, and laterally is the rounded capitellum, the two separated by an antero posterior ridge. The plane of the lower surface is not at right angles to the long axis of the bone. It has an outward tilt, forming the so called carrying angle of the elbow. This outward angulation of the forearm on the arm, with elbow extended and forearm supinated, varies between 2 and 16 degrees. The average is between 6 and 10 degrees.

The internal epicondyle is a prominent rounded projection frequently dimpled at its apex. The internal epicondylar ridge is regular, and forms a smooth concave line from epicondyle to shaft. A rare abnormality is the presence of the supra condylar process. This small hook like process projects

obliquely downwards from just in front of the epicondylar ridge. Usually a fibrous band joins this process to the internal epicondyle. Rarely the fibrous bridge is ossified, forming a tunnel which transmits the median nerve and the brachial artery or a branch thereof. This foramen is constant in many mammals.

The external epicondyle is flatter and less prominent than the internal and the epicondylar ridge above it has a slightly irregular convex outline.

The shadows of the trochlear and radial fossæ are superimposed on the olecranon fossa in an antero-posterior view to form a more or less triangular translucent area.

In the lateral view the forward curve of the lower end of the humerus is evident. Slight deviation from the true lateral position will mask this, the restoration of which is so important in the reduction of a fracture in this region. This view also shows the outlines of the capitellar and trochlear fossæ in front and the olecranon fossa behind. The cortical layers forming them can be traced from the shaft above till they fuse and then separate below. Occasionally this joint floor is perforated, forming the supratrochlear or intercondyloid foramen. The supratrochlear foramen is common in negroes, and is a frequent finding in prehistoric skeletons, the anthropoid apes, and many mammals.

### OSSIFICATION OF THE BONES OF THE ELBOW JOINT

At birth all the epiphyses of the elbow joint are cartilaginous. These ossify and fuse in the following order:

		Appear	Fuse together	Fuse with diaphysis.
Humerus	Ext epicondyle	10th-12th year	15th-17th year	17th-18th year
	Capitellum	1st-3rd year		
	Trochlea	11th year		
	Int epicondyle	5th-8th year	—	18th year
Ulna	Olecranon	10th-13th year	—	16th-20th year
Radius	Head	5th-6th year	—	16th-18th year
	Bicipital tuberosity	about 15th year	—	15th-16th year

The Capitellar Epiphysis of the humerus develops in the shape of a wedge, the apex directed inwards. As it grows it spreads and eventually forms most of the articular surface.

The Trochlear Epiphysis is small, forms only the medial part of the adult trochlear surface, and at first appears as two or more ossific centres.

A year or so before fusion with the diaphysis the external epicondylar, capitellar, and trochlear epiphyses join together. The internal epicondylar epiphysis fuses separately with the diaphysis.

The Olecranon Epiphysis, of the scale type, is very variable in its size and may appear at first as multiple ossific nodules. Even when it appears as a single scale, its deep surface is commonly notched, indicating the tendency to multiple ossification.

The Epiphysis of the Bicipital Tubercle of the Radius is relatively



uncommon. When present it is of the "scale" variety, appears about puberty and rapidly fuses with the main mass of the tubercle, which is of diaphyseal origin.

### HUMERAL SHAFT

The shaft of the humerus is cylindrical, and its principal radiographic feature is the deltoid tuberosity—a flat slightly roughened eminence on its outer surface near its middle. The diameter of the bone is narrowest just below this. The lower third of the shaft becomes progressively flattened from before backwards and presents a slight curve forwards.

In the upper third it widens out gently to merge with the head. Just below the head is the surgical neck, which however shows no radiographic delimitations.

### SHOULDER JOINT

**Standard Position.**—The systematic reproduction of a standard view is more difficult in the case of the shoulder joint than in others because of the varying



Fig. 225.—Adult shoulder joint in tangential and anteroposterior projections.

posture of the shoulder girdle in different individuals. For example, in round shouldered patients the scapula tends to point forwards compared with the normal, whilst in those of soldierly bearing the reverse obtains. Again variation in the centring point will result in considerable alteration in the relationship of the shadows of the bones. Similarly, rotation of the arm will alter the outlines of the humeral head.

For practical purposes it is perhaps best to adopt as the standard position that which the shoulder assumes at rest and in which it is usually bandaged immediately after an injury—namely the arm at the side and the forearm and hand on the chest (Fig 22a). With the patient on his back the head and shoulders are raised on an inclined plane to the extent of 10 to 15 degrees. The film cassette conveniently forms most of this inclined plane and sandbags and pillows the rest. The tube above is centred on the inner part of the humeral head. Another convenient method especially if the shoulder is seriously damaged is to take the radiogram with the patient erect against a vertical Bucky grid apparatus. The patient leans back on the grid with the tube in front of him.

External rotation of the humerus throws the greater tuberosity into relief and internal rotation brings the lesser tuberosity into view on the inner side of the head. A tangential view (Fig 22b) is sometimes valuable.

**Head of the Humerus**—In the standard view the humeral head is seen as a rounded mass. Most of the free outline is regular and smooth being formed by the articular surface. On the outer side is seen the irregular outline of the greater tuberosity and on the inner part close to the junction of the shaft the lesser tuberosity may be seen. Frequently the lesser tuberosity does not take part in the outline. The structure of the cancellous tissue of the head is irregular. Since it is not subject to pressure stresses it is not crystallised into regular striæ as in the femur. The humeral head is a not uncommon site of compact islands. Occasionally there occurs a line of cortical condensation at the top of the greater tuberosity. This has no pathological significance. A tent shaped epiphyseal scar is frequently observed at the base of the head.

The shadow of the humeral head is superimposed to a varying extent on those of the glenoid cavity and acromion process the amount of overlapping depending on the precise position in which the radiogram is taken.

**Scapula**—The general outline of the scapula varies according to its degree of forward inclination from the coronal plane. If the patient be very round shouldered the view obtained of the scapula in the standard position will be very foreshortened. A tangential view of the scapula taken with the arm raised is sometimes of value.

The glenoid cavity ovoid in outline with the apex upwards looks outwards and forwards and so a three quarter view is obtained of its outline. At its upper and lower articular margins there may be present small irregularities marking the origins of the long heads of the biceps and triceps respectively. The irregularity at the lower border is sometimes continued on to the neck of the scapula in the form of a small flat tuberosity.

The scapular spine is a prominent feature in the radiogram. Its outlines should be clearly visible including the flat triangular surface close to the vertebral border. Surmounting the spine is the curved acromion process the outer border of which often presents a series of blunt serrations.

The acromion process is very variable in shape, sometimes being gently curved, and at times being quite sharply angulated

The articular facet of the acromion is usually seen edge on in a radiogram. Its inclination may vary from the vertical to an angle of 45 degrees with that plane. In the angulated acromion the facet is usually angulated. Normally its margins should be smooth. Irregularity of its margins is usually the result of old trauma or osteo arthritic lipping.

As the hook like coracoid process points forwards and outwards it is seen foreshortened in the standard view. It can best be seen in profile by abducting the arm and angulating the central ray outwards. Occasionally an articular facet is present on its superior surface, for articulation with the conoid tubercle of the scapula.

The body of the scapula is irregularly triangular and shows considerable variation in the detail of its shape. On the superior border is to be seen the suprascapular notch, occasionally converted into a foramen by ossification of the suprascapular ligament.

The floor of the infraspinatus fossa in rare cases is deficient.

**Os Acromiale**—When the acromion epiphysis fails to unite, the resulting nodule of bone is called the os acromiale. It is a rare anomaly, and is more commonly bilateral than unilateral. One case seen by the author was bilateral. In it, a history of accident to one shoulder was given, and the condition was thought to be an ununited fracture of the acromion, until a radiogram of the other shoulder showed a precisely similar appearance. The accessory bone did not take part in the formation of the acromio clavicular joint.

**The Shoulder Joint**—Owing to the obliquity of the glenoid cavity a clear joint space is not as a rule visible and an estimation of the width of the joint space is difficult. In the position of rest the lower inner part of the humeral articular surface is in contact with the glenoid cavity, and the lower margin of the latter extends for a varying distance below the head of the humerus. When the arm is abducted, the lower inner articular margin of the head of the humerus descends, and occupies the same level as the lower margin of the glenoid cavity.

**Variations in the Standard Position**—(1) If the normal ray be centred medial to the shoulder joint, the glenoid cavity is seen more in profile, and the joint space can be better estimated. The coracoid shadow is elongated.

(2) If the centring point be below the joint, the acromion process is "spread out". This forms a useful view, therefore, for examination of that structure.

Centring above and external to the joint affords no advantages over the normal view.

# OSSIFICATION OF THE BONES OF THE SHOULDER JOINT

**The Upper End of the Humerus**—There are three centres of ossification for the upper end of the humerus as follows

	Appears
Head	1st year
Greater tuberosity	3rd year
Lesser tuberosity	5th year

This is the classic description given by *Gray Cunningham Morris Beclard Choyce Roberts and Kelly Cohn and Poland* state that all three developed from one epiphysis *R S Paterson* from his radiological studies came to the conclusion that in 50 per cent of subjects there is one epiphysis only and in the remaining 50 per cent there are two one for the head and one for the greater tuberosity. In none of his series of cases was a centre found for the lesser tuberosity.

These fuse together in the 6th year and join the shaft in the 20th year. Before the time of fusion with the shaft the united epiphyses fit like a cap on a conical shaped metaphysis.

In a radiogram the epiphyseal junction frequently casts a double shadow with a surprising gap between. This appearance is of importance as it may be mistaken for a fracture of the metaphysis (Fig 226).

**The Scapula**—The scapula is ossified from seven or more centres of which the following are constant: one for the body, two for the coracoid process, two for the acromion, one for the vertebral border, and one for the inferior angle.



FIG. 6.—Shoulder joint, child aged 10.

	Appears	Fuses
	End of month of fetal life	—
	10–18 months after birth	1st year
1 quick succession and in this order	14th–20th year	11th year
Body		
Body of coracoid process		
Root of coracoid (scale)		
Acromion body		
Inferior angle		
Tip of acromion		
Vertebral border		

The two acromial epiphyses fuse together before joining with the parent bone. Frequently the distal scale epiphysis is multiple at first. Failure on the part of the distal acromial epiphysis to fuse has already been noted as the explanation of the os acromiale. The base of the acromion is not formed

by these epiphyses, but by an extension outwards of the ossification of the scapular spine

The following epiphyses are inconstant

	Appears.	Fuses
Subcoracoid	10th-11th year	16th 18th year
This is a common epiphysis forms the upper third of the glenoid cavity		
Inferior glenoid	15th-17th year	23rd-25th year
This is a small epiphysis forming the lower part of the glenoid fossa		
Coracoid tip	15th-17th year	23rd-25th year

## CLAVICLE

The Clavicle is to a varying degree doubly curved, in S form. Its outline in a radiogram may vary markedly according to the degree of this curve, and to the exact disposition of the central ray. If this passes along the plane of the S curve of the clavicular shaft then the shadow of the shaft will be fairly straight. If the tube be angled to this plane, the S curve will be reproduced in the radiogram. Sometimes if the point of the shoulder be held forwards, part of the shaft of the clavicle may be seen almost end on, with the production of a cortical ring shadow. An almost constant feature is the downward projecting conoid tubercle, which affords attachment for the conoid part of the coraco clavicular ligament. The trapezoid ridge is rarely visible. A rare variation is a facet on the conoid tubercle, for articulation with the coracoid process of the scapula.

The Outer End of the Clavicle is expanded antero posteriorly, but this is not visible in a radiogram, in which only the very slight supero inferior expansion is visible. The outer end is tipped by the articular facet, which may be in a vertical plane or be inclined outwards or inwards from the vertical. The acromio clavicular joint shows a very varying joint space. This is dependent to some extent on the interarticular fibro cartilage. When this is absent the joint space is narrow. Commonly an incomplete cartilage occupies the upper half of the joint and in this case the upper part of the joint space is opened out in V formation. The outer end of the clavicle is usually a little higher than the acromion a relationship which should not be mistaken for a dislocation.

The Inner End of the Clavicle is considerably expanded. It articulates, through a fibro cartilage with the manubrium sterni and the inner part of the first costal cartilage. Its articular surface, concave from before back wards, is convex from above down, and extends on to the inferior surface. The inner end of the clavicle is projected considerably above the manubrium forming with its fellow the suprasternal notch.

**Ossification.**—The clavicle is the first bone in the body to ossify, a centre appearing for the shaft and outer end in the 5th or 6th week of foetal life. An epiphysis appears for the sternal end at about 18 to 20 years and unites about the 25th year.

## CHAPTER XVI

### NORMAL BONES AND JOINTS OF THE LOWER EXTREMITY

#### THE FOOT

**Standard Views**—There are four standard views

(1) **DORSO PLANTAR**—In this view the central ray should be normal to the dorsum of the foot, and consequently oblique to the film on the plantar surface

(2) **MEDIO LATERAL**

(3) **LATERO MEDIAL**

In the second view the metatarsals are more spread out than in the third otherwise there is little to choose between the two

(4) **PLANTO POSTERIOR**, to show the os calcis. The back of the heel rests on the film on the couch and the foot in strong dorsiflexion. The central ray is directed obliquely through the sole of the foot on to the film

#### THE PHALANXES

**Distal Phalanges**—The distal phalanx of the hallux is relatively large, and shows great variety in the details of its form. In the dorso plantar view its extremity is expanded in spade form with an irregular border from which varied excrescences may project.

The shaft is short and thick and expands out to fuse with the base. Frequently quite large tubercles project from the lateral and medial aspects of the shaft at the junction with the base.

The proximal surface of the base bears a biconcave articular facet with a median ridge, for articulation with the head of the proximal phalanx.

In the lateral view this phalanx is seen to have a broad base, and a shaft that tapers rapidly to the free extremity, which bears on its plantar aspect a roughened raised surface for the support of the pulp of the toe.

The distal phalanges of the other toes are diminutives of the first, and are at times fused with the mid phalanges.

The Mid-phalanges of the four outer toes have a bicondylar head and a biconcave basal facet. They become successively shorter and more cuboidal from the 2nd to the 5th toe, with progressive loss of form, until that of the 5th toe is merely a more or less cuboidal nodule of bone.

*Pfizzner* gives the percentage of fusion of middle and distal phalanges as follows

In the 5th toe in 36 per cent. of individuals  
In the 4th toe in 15 per cent. of individuals  
In the 3rd and 2nd toes in 0.75 per cent. of individuals

If fusion is present in the 4th toe, it is always present in the 5th and 6th.

**Proximal Phalanges**—These are all built on the same plan with a bicondylar head, a waisted shaft and a base bearing a concave articular facet. That of the hallux is large and substantially constructed. The others are relatively delicate. The lateral borders of the shafts may show small nodular projections similar to those seen in the fingers and marking the attachment of the tendon sheath.

Some outward obliquity of the proximal phalanx of the hallux is normal; an angulation increased in hallux valgus.

**Sesamoid Bones in the Toes**—These occur in the following percentages of cases according to *Pfitzner*:

1st metatarso-phalangeal joint	2 in 100 per cent
2nd metatarso-phalangeal joint	2 in 57 per cent
3rd metatarso-phalangeal joint	1 (tibial) in 16 per cent
Interphalangeal joint of hallux	1 in 54 per cent

**Sesamoids in the 1st Metatarso-phalangeal Joint**—Two are always present in the tendons of the flexor brevis hallucis. These sesamoids, it is important to note, are subject to many anatomical variations, the commonest of which is double sesamoid. Some of these double sesamoids are post-traumatic and some due to non-fusion of two ossific nuclei. Rarely the sesamoid may be represented by four or five tiny nodules of bone.

These abnormalities are commoner in the medial than in the lateral sesamoid, but may appear in either or both.

The sesamoids are frequently observed to be enlarged and deformed, but this is always pathological, such as in hallux rigidus. The deformity is then due to osteophytic formation.

## THE METATARSUS

The 1st Metatarsal, the shortest and strongest of the five, has a large rounded head covered distally and inferiorly by an articular surface. The head may present many varieties in its form and marginal osteophytic excrescences may occur in profusion. Although the latter are frequently symptomless and found accidentally in a radiogram, they probably always represent the first stages of a pathological state, such as hallux valgus, rigidus, etc.

The shaft is smooth in outline and shows on its inferior surface a ridge which commences about midway, and gradually becomes more prominent as it merges with the base at the infero-lateral angle.

The presence of this prominent ridge results in the proximal articular surface of the base being kidney-shaped or cuneate, with the apex downward and outwards. It articulates with a similarly shaped surface of the internal cuneiform. The importance of this configuration in the maintenance of the

medial longitudinal arch of the foot is obvious. The base of the 1st metatarsal occasionally articulates with that of the second.

The Four Outer Metatarsals are longer and more slender in construction. The head is largely covered with cartilage, the articular surface extending on to the plantar surface. Lateral tubercles are visible; these afford attachment to the lateral ligaments of the metatarso-phalangeal joint. The shafts taper from base to head and show a medial inclination relative to the bases. The recognition of the normally slender shaft, particularly at the neck, is of importance in connection with *Köhler's* disease of the metatarsal, in which the neck and shaft are thickened.

The bases of the 2nd, 3rd and 4th metatarsals are irregularly cuboidal. That of the 5th is triangular, so rendered by the prominent tubercle on its lateral aspect. Into this tubercle is inserted the tendon of the *peroneus brevis*.

The four outer metatarsals articulate by their bases with each other and also with the tarsus as follows:

1st metatarsal	with the three cuneiforms
2nd metatarsal	with the external cuneiform
3rd metatarsal	with the external cuneiform and cuboid
4th metatarsal	with the cuboid

## TARSUS

The Cuneiform Bones, as their name indicates, are wedge-shaped. The medial is the largest and least wedged of the three. Its medial surface is rounded, the apex of the wedge upwards. The distal surface, much larger than the proximal, is reniform.

The mid and outer cuneiforms are more typically cuneate, with their thin edges downwards. The middle is shorter than the other two, and the recess thus formed receives the base of the 2nd metatarsal. The cuneiform bones articulate distally with the four metatarsals and proximally with the scaphoid. The outer articulates with the cuboid.

The Cuboid.—This bone is irregularly pyramidal in shape, its base directed inwards. It articulates with the *os calcis* posteriorly, and its medial plantar corner projects under that bone.

The inferior surface presents an oblique groove for the tendon of the *peroneus longus*. This is visible in a lateral view, as is the ridge behind it, and the tuberosity at the outer end of the ridge. The tuberosity bears a convex facet on which glides the sesamoid or cartilage commonly present in the tendon of the *peroneus longus*. The outer surface, seen in profile in the dorso-plantar view, shows a notch—the commencement of the peroneal groove.

The cuboid articulates with four bones, the *os calcis*, the external cuneiform and the two outer metatarsals, and occasionally with a fifth, the scaphoid.

The Scaphoid, which is well seen in both the dorso-plantar and lateral views, has the form of a thick disc interposed between the astragalar head and



the three cuneiforms. Its anterior or distal surface is convex and bears three facets for the cuneiforms. The proximal surface is concave and articulates with the head of the astragalus.

The superior surface is seen in the lateral view to have a gentle convexity and is normally slightly roughened for ligamentous attachments. Osteophytic outgrowths are common on this surface close to the articular margins.

A prominent feature on the medial aspect of the bone is the tuberosity which is pointed downwards inwards and backwards. It affords attachment to part of the tendon of the tibialis posterior. Variations in this tuberosity are dealt with under the heading of the *Os Tibiale Externum*.

The scaphoid articulates normally with the astragalus and cuneiforms and occasionally with the cuboid.

**The Astragalus**—This is best seen in its entirety in a lateral view. Only the head is visible in a dorso plantar view of the foot whilst the upper half of the body is well seen in an antero posterior view of the ankle joint.

**THE HEAD** is smooth and rounded and articulates with the scaphoid. The under surface of the head bears two additional facets for articulation with the os calcis and inferior calcaneo navicular or spring ligament.

**THE NECK** is formed by a slight constriction or groove demarcating the head from the body. The upper surface of the neck frequently bears irregular nodules of bone. These are as a rule osteophytic. Occasionally a forward projecting spur takes rise from the upper surface of the neck. This the so called trochlear process is usually osteophytic but a developmental type has been described by *Thurstan Holland*. Below the neck is limited posteriorly by the middle calcanean facet.

**THE BODY** is irregularly cuboidal. Its upper surface bears the trochlear articular surface which also extends down on to the medial and lateral aspects of the bone. This surface is visible in both a lateral and an antero posterior view of the ankle joint especially in the latter.

The trochlear surface is broader in front than behind a configuration which prevents posterior luxation of the bone in the normal subject. It sometimes spreads on to the medial surface of the neck. This is common in races which habitually adopt the squatting position in which case it articulates with a similar facet developed on the anterior surface of the lower end of the tibia. It appears only occasionally in Europeans.

The inferior surface of the astragalus body is seen only in a lateral view in which its essential features may clearly be made out. Anteriorly is the middle calcanean facet articulating with the sustentaculum tali of the os calcis. Posteriorly is to be seen the larger posterior calcanean facet which articulates with the body of the os calcis. Between them is visible the deep oblique sulcus tali forming with the contiguous sulcus calcanei a tunnel—the sinus tarsi.

On the posterior surface the prominent feature is the external tubercle of the astragalus. This is varied in shape ranging from a flat rounded tubercle

to an elongated pointed process. If the latter it is frequently hooked down wards. Its relationship to the os trigonum is dealt with later under the heading of the accessory ossicles of the foot. The medial tubercle is small difficult to make out in a radiogram and is separated from the external by a groove in which runs the tendon of the flexor longus hallucis.

The angle of divergence of the neck of the astragalus is about 10 to 12 degrees inwards in Europeans. In the foetus and in the anthropoid apes it is much greater amounting to about 35 degrees. This angle is of importance in the estimation of the bony deformity in talipes equinovarus. For its demonstration a dorso plantar radiogram should be taken the centre point over the anterior part of the ankle joint with the foot in extreme plantar flexion.

The downward angulation of the neck of the astragalus is about 35 degrees in Europeans and less in lower races in which the foot is flatter.

The astragalus articulates with four bones the tibia fibula os calcis and scaphoid.

**The Os Calcis**—The lateral view shows this bone in its entirety. In this view it has an irregularly oblong outline with a narrower portion anteriorly.

The upper surface shows posteriorly a smooth slightly concave margin. Anterior to this the outline dips down at the posterior astragalar facet and separating this from the anterior astragalar facet in front is the sulcus calcanei forming with the sulcus tali the sinus tarsi. The sinus tarsi lodges the strong interosseous ligament and the nutrient vessels to both bones.

The anterior end of the upper surface is prolonged into a pointed edge or process. Usually blunt in outline in a lateral radiogram it may be sharp and represent a fused calcaneus secundarius (vide accessory ossicles).

The anterior outline is sinuous and articulates with the cuboid.

The inferior surface of the os calcis presents posteriorly a broad elevation—that of the posterior tuberosities. The anterior limit of this elevation is the commonest site for a calcaneal spur. In front of this is a slightly concave smooth outline becoming convex anteriorly and ending in a groove for the tendon of the peroneus longus.

The posterior surface is convex and smooth in its upper half which is separated from the tendo achilles by a bursa. The lower half is rough for the attachment of that tendon. The subcortical bone in the lower part of the posterior surface is frequently rather dense the result of the epiphyseal scar. The junction of these smooth and rough portions of the posterior surface is the site of the tendo achilles spur. This is quite a common abnormality but is probably always post traumatic following a tear or sprain in the tendon attachment. It may attain a considerable size and the distal part may be separate.

In the plantar posterior view the concave lateral surfaces of the os calcis are well shown in their posterior two thirds terminated by the rounded posterior surface of the heel.

In a lateral view the *lamellar structure* of the *os calcis* is clearly visible. Its regular arrangement is of considerable importance as after a fracture of any severity through the body of the bone the lamellar pattern is permanently deformed and gives what may be the only clue to an old healed fracture.

Three main bundles of lamellar striæ are present.

(1) Arising from the posterior calcanean facet and spreading out fanwise to end at the posterior surface and adjacent inferior tuberosity. This is the most pronounced bundle.

(2) A curved system of striæ arising from the anterior half of the inferior surface and sweeping backwards and upwards diverging somewhat posteriorly to end on the posterior surface. This system cuts across the first series at right angles to them in the posterior half of the bone.

(3) A smaller less defined fan system arising from the anterior astragalar facet and spreading out forwards and downwards to the cuboid surface.

The first and third systems are pressure striæ continuing downwards the vertical striæ in the astragalus while the second is a tension system.

### OSSIFICATION OF THE BONES OF THE FOOT

**Phalanges**—These are each ossified from two centres—one for the shaft and one for the proximal end or base.

	Appears	Fuses
Distal phalanges	Shaft 10th week	1 <sup>st</sup> 20th year
	Base 5th year	
Middle phalanges	Shaft 10th week	1 <sup>st</sup> 20th year
	Base 3rd year	
Proximal phalanges	Shaft 10th week	1 <sup>st</sup> 20th year
	Base 3rd year	

**Metatarsus**—The metatarsals also ossify from two centres—one for the shaft and one for the head—with the exception of the 1st metatarsal which normally has an epiphysis for the base and not for the head. Occasionally this bone has a second epiphysis for the head.

The diaphyseal centres appear in the 8th–9th week of intra uterine life and those for the epiphyses in the 3rd year. They fuse in the 17th–20th year. The occasional epiphysis for the head of the 1st metatarsal appears rather later about the 4th year.

**Tarsus**—The *os calcis* normally ossifies from two centres—one for the body and one for the posterior scle epiphysis. The former appears about the 6th month of intra uterine life. According to Kohler it commonly begins as two nuclei a lateral appearing first and rapidly fusing with a medial.

The posterior epiphysis appears in the 5th year. It may appear as early as the 3rd or as late as the 7th. It is commonly multiple beginning as two or even as three centres. These fuse together before fusing with the diaphysis.

The deep surface of the epiphysis is crenated or toothed the crenations interlocking with a similar irregularity on the diaphyseal side. In a radiogram this epiphysis casts a remarkably dense shadow compared with the rest of the bone,



FIG. 7.—Lateral view of the foot and ankle. Child aged 1.

frequently this is so marked as to raise the suspicion of osteochondritis. Comparison with the other side may serve to exclude the pathological condition.

The other tarsal bones ossify normally from single centres which appear at the following times

Astragalus  
Cuboid

6th month of intra uterine life  
9th month of intra uterine life

At birth therefore, the os calcis, astragalus and cuboid are visible in a radiogram.

External cuneiform  
Internal cuneiform  
Middle cuneiform  
Scaphoid

1st year  
3rd year  
3rd year  
3rd-4th year

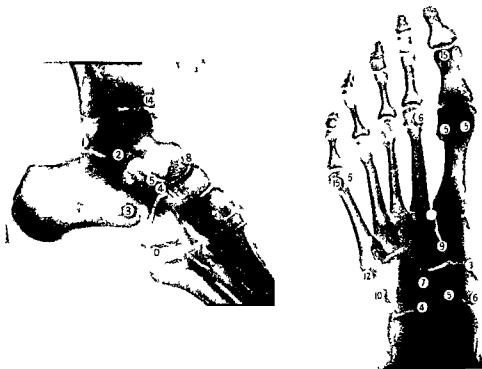
The scaphoid quite frequently begins to ossify from two centres which rapidly fuse. A double centre has also been described for the internal cuneiform.

### ACCESSORY OSSICLES OF THE FOOT

Many accessory ossicles in the foot have been described of which the following are more or less recognised as entities (Figs. 228 and 229).

(1) *Os Trigonum*—This ossicle the homologue of the semilunar bone of the hand and the representative of the primitive *os intermedium* is the most firmly established as an anatomical entity of all the accessory ossicles of the foot. Normally this embryological rudiment fuses with the lateral tubercle on the posterior surface of the astragalus. At one end of the scale of variations

is the ossicle fused to the astragalus in a relatively inconspicuous tubercle. At the other a completely separate ossicle lying in the angle between the astragalus and os calcis. It may then be of varying size and shape in size from 1 to 5 or 6 mm in diameter, in shape round or triangular. An inter



FIGS 28 and 29 Normal foot lateral and dorso-plantar projection and cutting the sites of sesamoids and accessory ossicles (1) Os trigonum (2) Os sustentaculum proprium (3) Calcaneus accessorius (4) Calcaneus secundarius (5) Os cuneum trochleæ (6) Os tubale externum (7) Cuneolus secundarius (8) Astragalo-scaphoid ossicle of Pirie (9) Os intercuneiforme (10) Sesamum peroneum (11) Os intermetatarsium (12) Os valanum (13) Os paracuneiforme (14) Astragalus secundarius (15) Common sesamoids (16) Rare sesamoid

mediate variety is seen in the form of an elongated lateral tubercle with a bulbous tip this latter representing the ossicle that has just fused and no more. This intermediate type is very susceptible to traumatic separation. *Kohler* has described fracture of a pointed lateral tubercle with in addition a discrete os trigonum. According to *Thurstan Holland* the os trigonum may very rarely be fused to the upper surface of the os calcis. The os trigonum is present as a separate ossicle in 7 per cent of subjects.

(2) **Os Sustentaculum Proprium**—This is described as a separate ossicle forming the posterior part of the sustentaculum tali. It has not been demon

strated radiographically and its existence is doubtful. It was first described by Pfitzner.

(3) *Calcaneus Accessorius* — This is said to occur as an epiphysis for the peroneal tubercle of the os calcis. Its existence is doubtful.

(4) *Calcaneus Secundarius* — This is a rare accessory ossicle. When present it occurs as a small irregularly quadrilateral bone lying in the angle between the os calcis, astragalus, cuboid and scaphoid. A more common appearance is a spur projecting from the os calcis into this angle. This represents a fused calcaneus secundarius. This spur may end in a flat surface articulating with the scaphoid.

All the above varieties are very rare. The usual finding is that of a clear space between the four bones mentioned.

(5) *Ossiculum Trochleæ* — This is said to occur on the outer side of the inferior calcaneo navicular (or spring) ligament. Its existence is doubtful.

(6) *Os Tibiale Externum* — This important ossicle is met with according to Pfitzner in 10 per cent of individuals. It is situated close to the tuberosity of the scaphoid in the angle between it and the head of the astragalus. It may vary from a nodule a millimetre in size up to an oval or reniform body a centimetre in length. It is more commonly bilateral than not, but as a rule it is larger on one side than the other.

There are three views as to its morphology.

(a) That it is a sesamoid in the tendon of the tibialis posterior.

(b) That it represents a separate ossific centre in the tuberosity of the scaphoid. One case of the author's supports this view, where the ossicle was too large to be a sesamoid and in which the tuberosity of the scaphoid was virtually absent and appeared to be represented by the ossicle.

(c) That it represents an old fracture of the scaphoid tuberosity.

Possibly all three types occur.

In a certain percentage of cases the presence of this ossicle, especially if large, is associated with pain and swelling over the tuberosity of the scaphoid even in the absence of any history of trauma.

(7) *Cuboides Secundarium* — This exceedingly rare bone has been recently described by Dwight and Schwalbe in the sole of the foot between the cuboid and scaphoid. It appears in a dorso plantar radiogram as a small circular shadow overlapped by the outer part of the scaphoid. It may be fused with the scaphoid or cuboid.

(8) *Astragalo scaphoid Ossicle of Pirie* — This is situated on the dorsum of the foot between the astragalar head and the scaphoid. In most cases it is probably merely an osteophyte separated from the adjacent margin of the scaphoid but is occasionally a true accessory ossicle.

(9) *Os Intercuneiforme* — This is very rare and occurs between the internal and middle cuneiforms dorsally.

(10) **Sesamum Peroneum** —This bone lies in the tendon of the peroneus longus where that tendon grooves the outer surface of the cuboid. It is very commonly present and is always bilateral. It may vary in size from a mere dot to an oval a centimetre or more in length and may be double.

(11) **Os Intermetatarseum** —This lies in the first interspace between the bases of the 1st and 2nd metatarsals. It is excessively rare as a separate ossicle and a little less rare in the form of a tiny spur fused to the base of the 2nd metatarsal.

(12) **Os Vesalianum** —This is an ossicle lying close to the tip of the basal tuberosity of the 5th metatarsal.

Two types have to be distinguished.

(a) An ununited epiphysis for the tuberosity. In this case the ossicle is large, has the shape of part of the tuberosity and is bilateral.

(b) A small ossicle close to the tip of the tuberosity, the latter being normally shaped. This is the true os vesalianum.

The Robert Jones fracture of the base of the 5th metatarsal must be distinguished from the former.

The following have been described but are doubtful entities.

(13) **Os Paracuneiforme** on the inner side of the foot in the angle between the scaphoid and internal cuneiform.

(14) **Astragalus Secundarius** on the dorsum of the foot between the astragalus and the lower end of the tibia. It is not uncommon and probably results from the detachment of an osteophyte.

(15) **Os Subtibiale** a nodule of bone close to the tip of the internal malleolus. *Fairbank* described such an ossicle arising from a separate centre of ossification for the tip of the internal malleolus but usually such a nodule is post-traumatic. Another view is that it is a sesamoid.

## JOINT CAVITIES OF THE TARSUS

These are six in number excluding the ankle joint. They are of importance in the spread of joint infections and can all be made out in either a dorso-lantar or a lateral view.

(1) **POSTERIOR CALCaneo ASTRAGALOID** —This is limited anteriorly by the interosseous ligament in the sinus tarsi.

(2) **COMMON CAVITY FOR THE ANTERIOR CALCaneo ASTRAGALOID AND ASTRAGALO NAVICULAR JOINTS**

(3) **CALCaneo CUBOID**

(4) **NAVICULO CUNEIFORM** —A complicated joint space common to the scapho-cuneiform articulations, the inter-cuneiform and those between the mid and external cuneiform and the 2nd and 3rd metatarsals. The anterior and posterior portions of this space communicate between the mid and external cuneiform bones.

(5) COMMON CAVITY BETWEEN CUBOID AND 4TH AND 5TH METATARSALS, extending forwards between the bases of the latter

(6) CAVITY BETWEEN INTERNAL CUNEIFORM AND 1ST METATARSAL

### LOWER ENDS OF TIBIA AND FIBULA, AND ANKLE JOINT

There are three standard views for the demonstration of these structures

(1) ANTERO POSTERIOR —The central ray through the joint space, the foot dorsiflexed to a right angle and inverted 15 to 20 degrees. The inversion is necessary to allow for the outward inclination of the antero posterior axis of the ankle joint

(2) MEDIO LATERAL }  
(3) LATERO MEDIAL } —The central ray over the joint space

**Lower End of the Tibia** —In the antero posterior view the shaft of the tibia broadens smoothly out into the expanded lower end. On the inner aspect is seen the internal malleolus smooth on its subcutaneous surface, and frequently rather irregular at its tip where the deltoid ligament is attached. Its deep surface is articular. Usually there is some indication in this view of the groove on the posterior surface of the internal malleolus—a groove which transmits the tendons of the *tibialis posterior* and *flexor longus digitorum*. The inferior surface is articular, and shows in this view a slight median ridge. It is continuous with the articular surface of the internal malleolus and also with a small facet on the outer surface. Occasionally in races which adopt the squatting position the articular surface is continued on to the front of the bone for articulation with a similar facet on the astragalar neck.

The outer surface presents a triangular depression above the small articular facet already mentioned. Into this depression the fibula fits, articulating below and being bound above by a strong interosseous ligament.

It is important to note that normally 'daylight' cannot be seen between these two bones. A gap of any size between them indicates a rupture of the interosseous ligament.

In the lateral views the anterior surface is rounded and often shows a slight ligamentous irregularity at its junction with the inferior surface. The inferior surface is concave. The posterior part of the lower end of the tibia is in the form of a blunt prominence in this view, and tends to overhang the anterior part. The internal malleolus is seen to be on a plane anterior to that of the external malleolus and is shorter than the latter.

**The Lower End of the Fibula** is visible in both views. In the antero posterior view its deep surface presents from above downwards a rough interosseous surface, a flat articular facet, and a ligamentous fossa just above the tip. The subcutaneous surface is smooth. Epiphyseal scars are commonly seen in both tibia and fibula.

**The Joint Space** should be clear and unobstructed in the antero posterior view, except that at times its middle section may be overlapped by the over



hanging posterior edge of the lower end of the tibia. In the lateral views the joint space is overlapped by the shadows of both malleoli. The average width of the joint space is 3 mm.

## OSSIFICATION OF THE LOWER ENDS OF THE TIBIA AND FIBULA

The epiphyses for these appear and fuse in the following order

Lower end of tibia	Appears	End
Lower end of fibula	1st year	18th year
	nd year	18th 0th year

Occasionally a second centre appears for the tip of the internal malleolus (*vide os subtibiale*)

The lower tibial epiphyseal cartilage has a slight sinuous curve viewed in the antero posterior position and this explains the double outline it frequently presents in the lateral view. At times also a double outline is seen in the antero posterior view in the medial half of the cartilage. This is due to a similar antero posterior curve in the cartilaginous plane. These appearances must not be mistaken for fractures.



## THE TIBIAL AND FIBULAR SHAFTS

The Tibial Shaft well seen in both antero posterior and lateral views tapers gradually from its upper end to its lower third where it gently expands into the lower end of the bone (Fig 230). This tapering is more evident in the lateral view. The cortex of the shin is remarkably thick in the middle third and in that section usually shows a slight convexity. The posterior surface is slightly concave. The subcortical trabeculation is often rather coarse in the mid third. The nutrient foramen is rarely visible.

**PLATYCNEMIC TIBIA**—In this rare anatomical variation the shaft is expanded from before backwards and compressed bilaterally. It has no clinical significance.

The Fibular Shaft is so variable in its form as to be difficult of detailed description. The lateral view shows it to be usually almost completely behind the tibia overlapping the shadow of the latter only at its lower end. In this view its anterior interosseous ridge is seen often showing mild irregularities. The shaft has a slight concavity forwards.

**COULTERED FIBULA**—This term is applied to a rare variation in which the shaft is so compressed as to be almost knife edged.

FIG 3) Normal adult tibial and fibular shafts lateral view

## THE UPPER END OF THE FIBULA

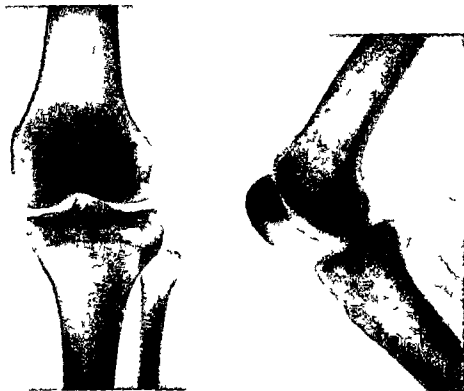
The fibular head varies considerably in size, position and shape. It is surmounted by an articular facet which faces upwards and forwards, the precise obliquity depending on the position of the fibular head relative to that of the tibia. If the fibular head be high it looks forwards and very slightly upwards and *vice versa*.

Occasionally the head is placed so low that it fails to articulate with the tibia in which case the facet is absent. Normally the head is placed posterolaterally to the outer tuberosity of the tibia. The best view of it is obtained in the lateral position.

## THE KNEE JOINT

With this will be described the upper end of the tibia, the patella and the lower end of the femur (Figs. 231 and 232).

**Standard Positions**—Four may be used—the antero-posterior, postero-anterior, medio-lateral and latero-medial. Two at least are essential in any case.



FIGS. 231 and 232.—Adult knee joint in antero-posterior and lateral views.

The two used as a routine are

(1) **ANTERO POSTERIOR**—The foot should point directly upwards. The central ray must be accurately centred over the joint. Unless this be done a clear view of the joint space will not be obtained.

(2) **MEDIO LATERAL**—The knee should be semi flexed the patient lying on his side and a small pad placed under the heel. The central ray must be centred over the joint space midway between the anterior and posterior surfaces of the limb. Centring too far forwards or backwards results in an oblique distortion of the shadows of the femoral condyles.

(3) **LATERO MEDIAL**—As No. 2 but with the tube below the couch.

(4) **POSTERO ANTERIOR**—This is used only to show the patella.

**The Upper End of the Tibia**—In the lateral view there is to be seen anteriorly the prominent tubercle of the tibia varied in shape but smooth in contour. The patellar tendon can be seen passing down to be attached to it. Above the tubercle the contour of the bone is smooth and its junction with the upper contour is rounded. The upper contour is complicated by the shadow of the tibial spine pointing upwards in a blunt peak. Below this the articular margins show as superimposed slightly concave lines. The posterior contour is rounded. The upper end of the tibia overhangs the shaft posteriorly a point clearly seen in this view.

In the antero posterior view the upper end of the tibia presents the two tuberosities lateral and medial the former being if anything more prominent. The superior contour is formed by the two articular facets. Their lateral concavity is evident in this view and their antero posterior concavity indicated by the anterior and posterior margins being visible as separate lines.

Separating the facets are the twin upward pointing peaks of the tibial spine. The medial is usually slightly higher and the points blunt. They become spiked in early osteo arthritis. The facets extend upwards on to the spine.

The internal architecture of the upper end of the tibia should be visible consisting largely of a longitudinal system of pressure striae. An epiphyseal scar is commonly present.

**The Lower End of the Femur**—In the antero posterior view the femoral shaft is seen to expand into a roughly quadrilateral mass the lower end. The lateral aspects of the condyles form the lateral contours. The medial extends higher than the lateral and ends above in the adductor tubercle. Frequently the lateral shows a double contour the posterior articular edge of the condyle being visible through the bone.

The inferior articular surface is smooth and saddle shaped.

The lateral articular margins are rounded off abruptly but smoothly into the lateral contours of the bone.

In the lateral view the condylar outlines are superimposed and present two curved lines. These lines form a blunt angle with the anterior surface of

the bone and commencing as a gentle curve increase in convexity backwards to form the prominent posterior portions of the condyles

Inside the two condylar lines is seen a third less regular U shaped line. The posterior half of this shadow is cast by the cortex of the floor of the intercondylar notch the anterior half represents the subcartilaginous cortex of the medial groove of the trochlear articular surface. The ends of this line blend above with the cortex of the shaft.

The Patella is best visible in the postero anterior and lateral views.

In the former it is seen through the shadow of the lower end of the femur, and has the shape of a rounded triangle apex downwards. It is in this view that a stellate fracture without separation is best seen.

In the lateral view the patella presents two borders upper and lower and two surfaces superficial and deep.

The superficial contour of the patella is smooth and convex becoming rounded off above into the upper border. Here it is frequently roughened for attachment of the superficial fibres of the quadriceps femoris tendon. This tendon should be visible being denser than the suprapatellar synovial pouch underneath it. The lower border is more pointed and running down from it is to be seen the patellar tendon. The deep surface of the patella is in its upper two thirds concave and articular. Below this portion and separated from it is a rough non articular surface.

The structure of the patella is best studied in the lateral view. Two layers of tension fibre are present. The superficial subcortical one is the more marked and is designed to take the stress of the quadriceps. A shorter less defined system lies below the articular cartilage. These two systems are buttressed apart by a pressure system spreading forwards fanwise from the lower part of the articular surface.

Disturbance of the normal internal architecture of the patella may give the clue to an old healed fracture.

**EMARGINATION OF THE PATELLA**—Under this name Kempson has described a concavity in the outer margin of the patella in its upper part bounded by a tubercle above and a spine below. It is an anatomical variation and is related to the mode of attachment of the tendon of the vastus externus.

Congenital absence of the patella has been recorded. A less uncommon abnormality is congenital outward dislocation of the patella.

**PATELLA BIPARTITA AND MULTIPARTITA**—In this variation the upper outer angle of the patella is discrete from the main mass. The small fragment may again be subdivided. It results from the failure of multiple ossific centres to fuse. It is usually bilateral and its bilateral nature serves to distinguish it from an ununited fissured fracture.

**The Fabella**—This is a small rounded sesamoid bone in the outer head of the gastrocnemius muscle. Its anterior surface is usually flat and faceted for articulation with the posterior surface of the external femoral condyle. It

varies in size its diameter being from  $\frac{1}{2}$  to  $1\frac{1}{4}$  cm. It is present in about 1.0 per cent of individuals. Very rarely one is also present in the inner head of the gastrocnemius. It must be distinguished from a loose intra articular osseous body from which its position usually serves to distinguish it. In the lateral view it lies behind the condyle separated from it by a few millimetres. A loose body in the posterior part of the joint lies closer to the condyle or is overlapped by the shadow of the latter. In the antero posterior view the shadow of the fabella is seen through that of the external condyle.

The fabella is as a rule bilateral and does not appear before the 19th to 15th year. It may show lipping and deformity in osteo arthritis of the joint.

**The Joint Space of the Knee** —This varies in width inversely with the age of the subject and directly with its size. In the young adult its width is in the region of 5 mm. The space is due entirely to the thickness of the articular cartilage proper. The semilunar cartilages take no part in maintaining it. The latter merely fill in the gap between the articular cartilages marginally and removal of a meniscus causes no narrowing of the space.

**Soft Tissues around the Knee Joint** —Anteriorly the quadriceps and patellar ligaments are clearly seen in the lateral view. Deep to the former is visible the suprapatellar synovial pouch. This is thrown into further relief if it be distended with fluid. Underneath the patellar tendon is a rhomboid space that occupied by the infrapatellar pad of fat. Mottling due to this fatty tissue is normal. In chronic villous arthritis this mottling is marked. Posteriorly the outlines of the heads of the gastrocnemius are apparent and their anterior borders mark the plane of the joint capsule. The semilunar cartilages cannot be made out in a plain radiogram consequently displacement of a meniscus gives no direct radiographic sign unless a bony spicule be also avulsed.

### OSSIFICATION IN THE REGION OF THE KNEE JOINT

The ossific centres in the region of the knee joint appear and fuse at the following times

	App. ara.	Fuses.
Fibular head	3rd-5th year	20th year
Tibial head	Just before or after birth	1st year
Lower end of femur	9th month	20th-22nd year
Patella	nd 5th year	
Fabella	12th-15th year	

**The Fibular Head** ossifies from one centre which appears between the 3rd and 5th years and fuses with the diaphysis at about the 20th year.

**The Tibial Head** also develops from one centre. At first round it gradually assumes an oval shape at the same time developing a nodular contour. In the early stages the outline of the adjacent metaphysis is convex and much wider than the epiphysis. Later the epiphysis takes on the adult shape with the tibial spine and the two tuberosities (Fig. 233).

About the 8th-10th year there develops a downward projecting tongue from the front of the epiphysis to form the tibial tubercle. A recognition of the varied forms which this epiphyseal tongue may take is of great importance in determining the presence or otherwise of Schlatter's epiphysitis. In this respect it should be noted that the lower part of the epiphyseal tongue may have a separate ossific centre. mere fragmentation of the epiphysis therefore does not necessarily indicate an epiphysitis. Again the epiphysis is frequently separated from its diaphyseal bed by a space and has the appearance as if it had been lifted from its bed by the pull of the patellar tendon. This is a normal appearance and must not be mistaken for a traumatic lesion. In the study of this epiphyseal tubercle it is quite essential to take a control radiogram of the other side and even then a correct decision can sometimes be arrived at only by a consideration of the local clinical signs.



FIG. 933.—Knee joint, lateral view. Child aged 10.

**Patella**—The patella ossifies normally from one centre. The centre is very variable in its time of first appearance. As a rule it is first visible during the 2nd or 3rd year but may be delayed as long as the 6th. Rarely two or more centres are present. If these do not fuse a bi- or multipartite patella results.

In the early stages of its ossification the patella presents a remarkably nodular outline in a radiogram reminiscent of the outline of a mulberry urinary calculus. As ossification reaches its completion this irregularity gradually disappears. Ossification is usually complete about the 10th year.

**Lower End of Femur**—This ossifies from one centre. This centre is of obstetric interest inasmuch as its presence is generally accepted as proof of the maturity of the fetus. It normally appears during the ninth month of intra uterine life but according to Kohler may be delayed as long as the 3rd month of extra uterine life. At birth it is about 3.5 mm in diameter. As growth progresses it assumes an oval shape and later gradually takes on the adult bicondylar form. In its earlier stages it presents the irregular or mulberry contour normally seen in so many of the developing epiphyses.

The contour of the adjacent metaphysis is convex and its margins rather pointed in the earlier stages of the epiphyseal development. The presence of

a sharp spike at these margins is not uncommon but sometimes results from a degree of minor trauma such as a mild epiphyseal strain

The knee being the growing centre for the lower limb the lower femoral epiphysis fuses late uniting between the 20th and 22nd years

### THE SHAFT OF THE FEMUR

The femoral shaft appears smooth and cylindrical with a thick well developed cortex Although in cross section it has the form of a rounded triangle this is not evident in a radiogram

In the antero posterior view it is quite straight and narrowest at its middle where its cortex is very thick It increases gradually in thickness as it approaches the lower extremity the cortex becoming thinner at the same time It joins the lower end at a slight angle Towards its upper end the shaft also widens to a lesser degree than below

In the lateral view the shaft shows a gentle convexity forwards The posterior contour may be very slightly irregular but as a rule no trace of the *linea aspera* is visible In the anomaly known as the pilastered femur the *linea aspera* is very prominent and shows in a radiogram (*vide infra*)

**PLATYMERIC FEMUR**—In this rare anomaly the shaft of the femur is flattened somewhat from before backwards below the trochanters Its cause and significance are unknown

**PILASTERED FEMUR**—This is also a rare abnormality in which the whole bone is heavily sculptured with the normal ridges accentuated In addition the articular margins of the head are lipped The pilastered femur is found in individuals of great muscular development

### THE HIP JOINT

There are four standard antero posterior positions for radiographing this joint In all the central ray passes through the joint

(1) **NORMAL**—Feet together toes pointing upwards This gives the most satisfactory general view and should always be used in routine work (Fig 234)

(2) **INTERNAL ROTATION OF THE FEMUR**

(3) **EXTERNAL ROTATION OF THE FEMUR**

(4) **ABDUCTION OF THE FEMUR**

The appearances in the normal position will be described and the variations in the other positions briefly outlined subsequently

The lateral view of the femoral neck latero medial or medio lateral with the leg abducted is used to check the insertion of a peg in fracture of the neck

**The Upper End of the Femur**—On following the femoral shaft upwards it is seen to widen gently and end in the two trochanters On the inner side the lesser trochanter presents a smooth round prominence The greater trochanter

has a roughly quadrilateral outline with blunt angles. The tip of the trochanter lies above the contour of the femoral neck, and the digital fossa can be made out between the superimposed shadows of neck and trochanter.

The femoral head has a circular or slightly oval contour interrupted medially for about half an inch by the depression of the fovea capitis. The inner half of the femoral head is overlapped by the shadow of the acetabulum. On rare occasions the articular surface of the head extends on to the outer surface of the neck, forming the *eminentia articularis colli femoris*.

**STRUCTURE OF THE UPPER END OF THE FEMUR**—The architecture of this is clearly visible in a radiogram. The cancellous lamellæ are so arranged as to withstand the pressure of weight bearing.

(1) In the head proper a series of bony lamellæ converge to a dense wedge or triangle (Ward's triangle) of cancellous tissue in the middle. This central wedge represents the epiphyseal scar.

(2) From this scar a fan of lamellæ spreads downwards to join the side of the neck. These stræ are strongest and best marked on the inner side, where the cortex of the neck is thick and strong.

(3) A third series of stræ extends from the region of the digital fossa to the lesser trochanter and

(4) A feeble band crosses the base of the great trochanter. These two form the uppermost of a series of arched lamellæ extending across the upper end of the shaft.

(5) The *calcar femorale*, or femoral spur. This is a strong plate of bone embedded in the femoral neck. It takes its origin from the cortex just in front of the small trochanter, and reinforces the vertical lamellæ in the inner side of the neck. It is well seen in a radiogram.

**TRANSLUCENCIES IN THE FEMORAL HEAD**—Two translucent areas are normally visible in the femoral head, on the inner and outer aspects close to the neck. They are bounded medially by the acetabulum, and below by the



FIG. 234.—Adult hip joint. Antero-posterior view.



**epiphyseal scar** They result from a thinner mass of bone to be penetrated by the X ray on the periphery of the femoral head and must not be mistaken for pathological translucencies

**The Acetabulum**—This cavity cup shaped for the reception of the femoral head is seen obliquely in the normal view Starting at the upper outer margin seen in profile three outlines can be traced downwards

(a) The innermost is a dense semicircular line following the contour of the femoral head and represents the cortex of the floor of the acetabulum In its upper part it is separated from the femoral head by a space of 4-5 mm in the adult—the joint space With increasing age this space narrows from absorption of cartilage Opposite the fovea capitis this line bulges inwards slightly and is often broken Below this it ends in the cotyloid notch In its inner half this line is crossed by another curved line which sweeps from the brim of the pelvis and represents the cortex of the pelvic wall

(b) The middle line concave outwards is cast by the anterior lip of the acetabulum and also disappears at the cotyloid notch

(c) The outer line formed by the posterior margin of the acetabulum is more or less straight roughly bisects the femoral head and ends below in the outer contour of the ischium

**SHENTON'S LINE**—This line is plotted out by following outwards the inferior border of the shadow of the ascending pubic ramus and continuing outwards and downwards along the medial border of the femoral neck It forms a regular curve both in the normal position in adduction and abduction Its regularity is disturbed in a wandering acetabulum coxa vara coxa valga or upward displacement of the femoral head from any cause and it forms an accurate test for the presence of such conditions

**Variations in the Radiographic Appearance in the Three Other Positions**—In internal rotation of the femur the neck is seen in its fullest extent and the lesser trochanter almost disappears behind it

In external rotation the neck is foreshortened its shadow partly overlaps that of the great trochanter and the small trochanter comes well into profile

In abduction the appearances in the above are exaggerated and the trochanter and neck are almost in line

In all the above views Shenton's line retains its regular arch

In the abducted latero-medial and medio-lateral views the femoral shafts trochanters and neck are brought more or less into line

## OSSIFICATION IN THE REGION OF THE HIP JOINT

Three epiphyseal centres take part in the formation of the upper end of the femur one for the head and one for each trochanter The neck is diaphyseal

**The Epiphysis for the Head** appears during the 1st year and fuses with the neck about the 18th to 20th year At first a rounded nodule of bone by the

3rd year it has assumed a semicircular outline, the *juxta metaphyseal surface* being more or less straight and horizontal. By the 10th year the bone has the appearance as though the inner and lower part of the head were diaphyseal in origin, but as growth progresses the epiphysis grows inwards and downward to cap the inner portion of the neck, and to form the whole of the articular surface. The epiphyseal cartilage is thus eventually somewhat domed or tented, and after fusion of the epiphysis leaves a similarly shaped epiphyseal scar.

The Epiphysis for the Great Trochanter appears about the 3rd to 5th year. Several successive discrete centres may form in line, and these soon fuse to form a single nucleus. The epiphyseal cartilage shows as a straight line inclined upwards and inwards at an angle of about 45 degrees. Soon the epiphysis assumes a pyramidal shape to form the upper and outer half of the trochanter. The base is diaphyseal in origin. Fusion occurs between the 18th and 20th years.

The Epiphysis for the Small Trochanter is variable in its time of appearance, from the 8th to the 14th year. The 12th year is the commonest time. It is a cupped scale epiphysis, and fuses about the 18th to 20th year.

The Acetabulum.—The three primary centres for the innominate bone grow down into the cartilaginous acetabulum in the form of blunt angular segments and together form that cavity in the following proportions:

- (1) Iliac centre,  $\frac{2}{3}$ ths
- (2) Ischial centre,  $\frac{1}{3}$ ths
- (3) Pubic centre,  $\frac{1}{3}$ th

Separating these three processes is the Y shaped cartilage.

At the ages of 1 to 3 all three limbs of this cartilage may be seen, but that between the ischium and pubis soon becomes obscured in an antero-posterior radiogram by the overlapping of these developing elements. The two upper more or less horizontal limbs remain visible until fusion. The contour of the iliac segment of the acetabulum is normally nodular and irregular from the 5th up to about the 12th year.

About the 12th or 13th year small centres of ossification appear in the limbs of the Y shaped cartilage. That between the ilium and pubis is the best defined, and forms the anatomical os acetabuli. About the 18th year the os acetabuli fuses with the ilium and pubis and, shortly after, fusion takes place in the other two limbs. The term os acetabuli is also applied to a small ossicle lying close to the upper outer margin of the acetabulum.

#### BIBLIOGRAPHY

- ASHURST, J., *International Encyclopedia of Surgery*, London 1882.  
 BUCHANAN, A. M., *Manual of Anatomy*, London 1937.  
 COHEN, I., "Normal Bones and Joints," *Annals of Roent* II, New York 1924.  
 CUNNINGHAM, D. J., *Textbook of Anatomy*, Oxford 1937.  
 DWIGHT, T., "Variations of the Bones of the Hands and Feet," *Clinical Atlas*, Philadelphia, 1907.

- FAIRBANK, H A T, *Arch Radiol Electrol*, 1922-3, XXVII, 238  
 FRAZER, J E, "Anatomy of the Human Skeleton," London, 1933  
 GRAY, H, 'Anatomy,' London, 1938  
 HARRIS, H A, *Brit J Radiol*, 1931, IV, 622, "Bone Growth," Oxford, 1933  
 KEMPSON, F C, *J Anat Physiol*, 1901-2, XXXVI, 419  
 KOHLER, A, 'Roentgenology,' London, 1935  
 MORRIS, H, "Treatise on Anatomy," London, 1915  
 PATTERSON, R S, *J Anat*, 1929, LXIV, 28  
 PFITZNER, W, *Morphol Arbeit*, 1891-6, I-VI  
 PIERSOL, G A, 'Human Anatomy,' Philadelphia, 1930  
 POLAND, J, 'Skiagraphic Atlas,' London, 1898  
 QUAIN, J, "Anatomy," VI, 1, London, 1915  
 ROBERTS, J B, and KFLY, J A, 'Treatise on Fractures,' Philadelphia 1921  
 SCHWALBE, G, *Zeitschr f Morphol u Anthropol*, 1910, XIII  
 SCUDDER, C L, 'The Treatment of Fractures,' Philadelphia, 1938  
 SHENTON, E W H, "Disease in Bone and its Detection by the X Rays, London, 1911  
 SPALTEHOLZ, W, 'Hand atlas of Human Anatomy,' Philadelphia, 1932  
 THURSTAN HOLLAND C, *Arch Radiol Electrol*, 1921 XXVI, 105

## PART THREE

### SECTION I (*continued*)

## THE NORMAL SPINE AND PELVIS

BY

PETER KERLEY, M.D., F.R.C.P., F.F.R., D.M.R.E.

### CHAPTER XVII

#### THE NORMAL VERTEBRAL COLUMN AND PELVIS

THE NORMAL spine consists of 33 vertebrae grouped according to their situation as follows: 7 cervical, 12 dorsal, 5 lumbar, 5 sacral and 4 coccygeal. The sacral and coccygeal groups fuse in the adult to form two bones, the sacrum and coccyx, while the other groups remain separate throughout life. The separate vertebrae will be considered first.

#### OSSIFICATION

The essential parts of a vertebra are the body and the arch. These ossify from three primary centres: one for the body and one for each side of the arch. Ossification of the arches begins in the seventh week of foetal life in the upper cervical vertebrae and spreads rapidly downwards. Ossification of the bodies begins two or three weeks later in the mid-dorsal region and spreads upwards rather more rapidly than downwards.

At birth the two arches are still separate from each other and from the body of the vertebra. The arch centres fuse between one and seven years. The fusion between the arch and body takes place in the cervical region in the third year and in the lumbar region in the sixth year. In or about the age of puberty five secondary centres appear, viz. one for each transverse process, one for the spinous process and one each for the superior and inferior rims of the vertebral body. Normally all these epiphyses are firmly united at the age of 25 years.

The above description applies to all the vertebrae except the first, second and seventh cervical. The atlas is ossified from only three centres and complete fusion of these occurs fairly early in life. The axis ossifies from five primary centres: one from the body, two for the arch and two for the odontoid process. Secondary centres develop for the tip of the odontoid at 2 years and for the inferior rim of the body about puberty.

The seventh cervical vertebra is peculiar in having an extra primary centre for each costal process.

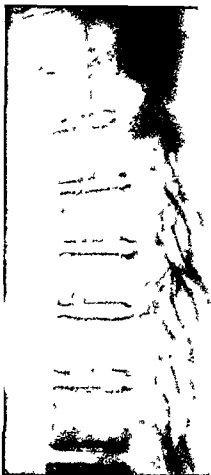


FIG. 235.—Lateral dorsal spine of an adult showing the compact bony rim of the bodies clearly.

(a) two cartilage plates which lie flat on the cancellous bone of each body (b) a structure known as the annulus fibrosus which is a dense fibro cartilaginous ring attached to the rim of each body and (c) the nucleus pulposus a soft oval gelatinous mass lying in the centre of the annulus fibrosus. It is obvious that the integrity of a structure like this can only be maintained if the cartilage plates are adequately pro-

### THE INTERVERTEBRAL DISCS

Until comparatively recent times the spine was regarded as a semi rigid column with a very limited range of movement. We now know the fallacy of this idea, and recognise the importance of the intervertebral discs which are really a series of strong joints permitting a wide range of movement. Unlike other joints of the human body, the joints between the vertebral bodies are subject to a constant and heavy strain, and nature has adapted them accordingly. There are three chief differences between the intervertebral joints and other joints: (1) there is no true joint space, (2) the joint cartilage is superimposed directly on cancellous bone, and not on compact bone, and (3) there is a semi fluid gelatinous nucleus which can expand and contract and which constitutes the essential pivotal point of an intervertebral joint.

There are three essential parts in a joint between two vertebral bodies



FIG. 236.—Antero posterior view of a lower dorsal vertebra in a child. The bubble effect is particularly well shown.

ected and if the annulus fibrosus is firmly attached to an unyielding base. To meet this need a dense compact bony rim is formed on the superior and inferior parts of the body (Fig. 235). The development of this rim from secondary centres produces curious X-ray appearances. On the surfaces of each vertebral body in children there are a number of grooves radiating in cart wheel fashion from a central point. These grooves are deeper, longer and more numerous towards the anterior part of the body. Each groove contains hyaline cartilage in which develops one of the small secondary centres for the bony rim, i.e. the bony rim of a vertebra is developed from multiple centres and not from a single centre as is commonly taught. In regions like the sacrum where the vertebral bodies eventually fuse and there is no need for a stout bony rim these grooves are slight and shallow and may not number more than two or three. In the dorsal region where there is most movement the grooves are very deep and there may be as many as seventeen or eighteen. In both antero-posterior and lateral views of a child's spine these grooves are clearly visible. In the antero-posterior view they appear as a row of bubbles on the superior and inferior margins of the body (Fig. 236) and in the lateral view as translucent areas between layers of fairly dense bone.

### VARIATIONS IN THE CHILD'S SPINAL COLUMN

The lateral radiographic view of a child's spine shows remarkable variations which are not constant for age or sex or race. In infants the vertebral bodies are bobbin-shaped and while some appear to be completely ossified others show a large anterior notch. Up to the age of 1 year the degree of ossification appears to be rather haphazard. There is often a deep narrow slit running through the middle of each body (Fig. 237). It is now generally accepted that this slit represents the channel of a diploic vein and is analogous to the diploic veins in the skull. In many cases there are deep notches in the anterior parts of the bodies, this notching producing a step like effect (Fig. 238). In each of these notches an ossified nucleus for the bony lip of the body appears

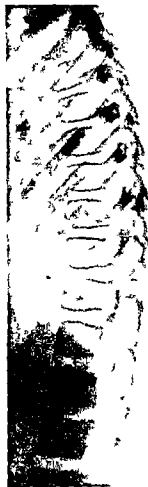


FIG. 237.—Normal lateral spine of child aged 10 years. lateral view. The slit like pleural vessels are well seen.



FIG. 38—Lateral view of a lower dorsal vertebra showing the steeple effect.

turbance of normal ossification. This latter view is at the moment a minority one.

### THE X RAY APPEARANCES OF THE ADULT SPINAL COLUMN

In the antero posterior view of a vertebra we see clearly the body with the shadow of a spinous process projected through the middle of it. This spinous process belongs to the vertebra immediately above except in the case of the fifth lumbar vertebra. On each side of the body the transverse processes stick out at right angles. The pedicles in this view appear as oval or circular rings. Above and below the pedicles are the superior and inferior articular processes. Generally speaking a vertebra is always as big as or bigger than the one immediately above it. In the cervical region the pedicles are oval and in the lumbar and dorsal region they are circular. In adults the medial aspect of the pedicles is always convex but in children there are considerable variations and it may be concave. The distance between the pedicles is greatest at the cauda equina and at the cervical and lumbar enlargements. It is least in the mid thoracic region. Alterations in the shape of the pedicles or in the interpedicular distance may have great diagnostic significance.

later i.e. we are seeing end on one of the centres for the bony rim to which is attached the annulus fibrosus. The time of appearance of these ossific nuclei varies very much. As a rule they are first seen between the ages of 10 and 18 years but they have been recorded as early as 6 years and in many cases they are never seen at all. The frequency and extent of these variations have led some workers to question whether this step like effect is in fact normal and it has been suggested in some quarters that it is an acquired disturbance.



FIG. 39—Normal to antero-posterior view of spine of child aged 10 years.

In the lateral view we see the bodies of the vertebræ as dense rectangular masses, behind them the pedicles separating the intervertebral foramina, and behind these again the spinous processes. The direction of the intervertebral foramina varies in different parts of the column. In the cervical region the foramina run obliquely, in the dorsal region more laterally, and in the lumbar region they run in a true lateral plane. Any of the secondary centres of ossification may remain separate.

### VARIATIONS IN THE ADULT SPINAL COLUMN

Trivial congenital malformations are relatively common in the vertebræ, and although these cannot properly be considered as normal the fact that they are symptomless and innocuous warrants their inclusion in this section. Incomplete fusion of the arch of the atlas is relatively common. This deformity is best seen in oblique views or in stereoscopic lateral views. Synostosis between the atlas and occiput is rare. Occasionally there is a transparent fissure between the odontoid process of the axis and the anterior tubercle of the atlas, this in the antero posterior view gives the impression of the odontoid being split in two. The spinous processes of the second, third, fourth and fifth cervical vertebræ show marked irregularity. The second, of course, is very big and long, while the next three are very short. All four, however, are frequently bifid. Persistence of the epiphysis of the sixth cervical spinous process has been described by Kohler and closely resembles a fracture.

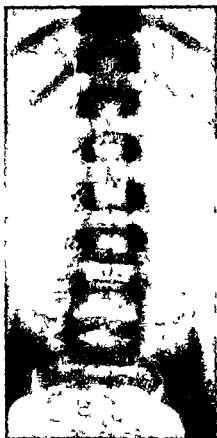


FIG. 240.—Normal antero posterior view of the lumbar spine of a child aged 10 years.

The transverse processes of the seventh cervical vertebra are much longer than the transverse processes above, and sometimes than those below. These processes run at an acute angle to the transverse processes of the first dorsal vertebra. Not infrequently they are of unequal length on either side, and care should be taken that the long one is not diagnosed as a cervical rib. If, however, a transverse process of the seventh cervical vertebra projects well



beyond that of the first dorsal and is also downward turned and pointed it may represent a fused cervical rib continued as a fibrous band to the middle of the first true rib. This band may produce pressure. Persistence of the secondary epiphysis for the transverse processes is often seen. Incomplete fusion of the arch of the seventh cervical vertebra is not uncommon and occasionally the first dorsal vertebra is similarly affected.

From a study of over 10 000 chest radiograms the writer estimates the frequency of this anomaly at 1 in 600. *Newman* once observed such incomplete fusion of the arch of C7 in a patient with Raynaud's disease. It is probable that this was a pure coincidence.

The only common variation seen in the dorsal vertebrae is persistence of the secondary centres for the transverse processes. Rarely persistence of one of the rim centres is observed in the lateral view. This is seen as a small well defined triangular piece of bone apparently separate from the superior or inferior edge of the body. The twelfth dorsal vertebra is often large and very similar to a lumbar vertebra. When this is so the fifth lumbar vertebra is usually sacralised to some extent. In such cases the twelfth ribs are missing. Persistence of the secondary epiphyses of the transverse processes of D12 and L1 is very common. Absence of the spinous process of the twelfth dorsal vertebra is a very rare malformation described by *Köhler*.

The chief malformations seen in the lumbar vertebrae are irregularity of the transverse processes. These vary considerably in size and not infrequently their secondary epiphyses remain ununited. So called sacralisation of one or both transverse processes of the fifth lumbar vertebra is a well known anomaly. The affected transverse process or processes are very wide



FIG 241 Anteroposterior view of normal adult dorsal spine

and thick and are completely fused to the sacrum or form a joint with the transverse processes of the first sacral vertebra. It is very doubtful if this disturbance of ossification should be included here as it is so often associated with backache and sciatica. The same remark applies to spina bifida occulta of the fifth lumbar vertebra which is also common and believed by many to be associated with minor nervous disorders such as nocturnal enuresis.

## THE SACRUM AND COCCYX

The sacrum is composed of five separate vertebrae which fuse together to form one large bone. It is shaped like an inverted triangle base upwards. Its dorsal surface is rough and uneven while its pelvic surface is smooth. Like other vertebrae there are three primary centres of ossification at birth for each sacral vertebrae viz one for the body and one on each side for the lateral processes. Secondary centres appear on the upper and lower surfaces of each body at puberty these fuse with the bodies and the bodies fuse with each other between the ages of 18 and 20 years. Other secondary centres appear on the ends of the costal and true transverse processes at the age of 18 years. It is usually impossible to demonstrate any of the sacral secondary epiphyses by radiography.

In an antero posterior view of the sacrum we see the constituent parts clearly. The body or central part because of the fused spinous processes behind appears much denser than the lateral masses. Between the body and the lateral masses on either side are the four sacral foramina. In rare cases the sacrum and coccyx are fused and there appear to be five sacral foramina.

The spines of the sacral vertebrae are usually fused to form a thick bony ridge but as the lower two sacral vertebrae have no true posterior arches the rough spine stops short at the level of the fourth foramina and from it run down two blunt bony projections known as the sacral cornua.



FIG. 24.—Normal lateral view of the lower lumbar spine.

A great variety of congenital anomalies occur in the first sacral vertebrae. Incomplete fusion of the arch is very frequent and often associated with spina bifida (clefts of the fifth lumbar vertebra). This deformity may be associated with minor nervous disturbance such as nocturnal enuresis or even with incontinence. Minor malformations or even absence of the first sacral spinous process are very common and are of no significance. Occasionally the first sacral vertebra tends to be lumbar in type i.e. one or both of its lateral masses

remains free and forms an independent part of the sacro iliac joint. Rarely the centrum may also be separated and a disc pressed between it and the second sacral body.

The coccyx like the sacrum, has the shape of an inverted triangle and is usually made up of four incomplete vertebrae fused together. The first vertebra which articulates with the sacrum is the largest and possesses a pair of processes projecting upwards known as the coccygeal cornua. There are usually well marked transverse processes for the first and second coccygeal



FIG. 143—Normal adult female pelvis

vertebrae. Considerable variations occur in the number and size of the lower coccygeal vertebrae. In most individuals there are only three which appear to be little more than rudimentary bone nodules, but as many as six have been described. The direction of the coccyx in the pelvis also varies enormously in different individuals. Such differences may be congenital or may be acquired during parturition and are often wrongly attributed to direct trauma. Unless an obvious gross lesion is present, it is usually impossible to tell by radiography whether the coccyx has been injured or not. Fortunately the debatable question of normal or traumatic displacement of the coccyx has not yet reached the law courts.

## THE PELVIS

The bony pelvis is composed of two hip bones, the sacrum and the coccyx. Each hip bone is composed originally of three distinct bones—the ilium, the ischium, and the pubis. In the adult these three bones are fused together at the acetabulum. Ossification occurs from three primary and several secondary centres. The primary centres appear in the third month for the ilium, in the fourth month for the ischium, and in the fifth month for the pubis. The secondary centres which appear about puberty are as follows: two or three for the triradiate strip at the acetabulum, one or two for the iliac crest, one for the anterior inferior spine, one for the ischial tuberosity, and one for the symphyseal part of the pubis.

The anatomical variations of the hip bones are few and seldom give rise to radiological difficulties. The epiphysis of the anterior inferior spine occasionally remains ununited. The ilium varies considerably in thickness and not infrequently linear areas of translucency are seen running across it. These linear markings are caused by nutrient vessels. Compact bone islets are rarely seen in the hip bones. Ill-defined translucent areas due to gas in the bowel are easy to identify and can be displaced by compression.

The size and shape of the normal female pelvis are of the greatest importance in obstetrics. They are dealt with in Vol. I, Part 5.

## PART THREE

### SECTION II

## THE GENERAL PATHOLOGY OF BONE

BY

S L BAKER, PH D M Sc MRCS LRCP

### CHAPTER XVIII

## THE GENERAL PATHOLOGY OF BONE

### INTRODUCTION

BONE HAS one outstanding peculiarity which is not found in the other tissues of the body—it is composed of well defined fragments deposited at different times during the life of the individual. The whole skeleton as was first pointed out by *von Ebner* (1875) is made up of a mosaic of microscopic fragments this structure being produced by continued alternations in the processes of bone erosion and deposition. These processes are very active during the period of growth when great changes in the size and shape of the bones are in progress but they continue in a less active form after the growth period and even into old age. The fact that the bones although mechanically stable and rigid are subject to continuous microscopic change was realised as soon as an adequate histological study of their structure had been made. The normal mechanism of bone erosion and deposition during the growth period was studied extensively by *Koelliker* (1873) and *Pommer* (1885) pointed out that these changes continue in a less active form even into old age.

In recent years these same facts have been studied from a different angle and work on calcium metabolism has shown that there is normally throughout life a continuous loss of calcium from the skeleton—corresponding to the continuous process of bone erosion by osteoclasts pointed out by *Pommer*. This calcium loss is normally made good by the deposition of fresh bone during the growth period bone formation exceeds erosion in adult life the two processes are approximately balanced in old age the process of bone formation lags behind that of bone erosion and a senile osteoporosis results.

It is well established that in addition to age variations the balance between

erosion and deposition is influenced by stresses and strains on the bone, with the result that bone tissue tends to be distributed along the lines of force and thus arranged to the best mechanical advantage

In some cases our knowledge of the mechanisms controlling bone structure has been derived from the study of normal bones, but in many cases it is derived from the study of pathological changes which result from the disturbances of mechanisms whose existence would otherwise have remained unsuspected. This is the case in such conditions as acromegaly and hyperparathyroidism, studies of which have revealed the important influences of the pituitary and the parathyroid glands on skeletal structure. The part played by vitamin D in the normal mechanism of bone calcification could only have been discovered by the study of rickets and osteomalacia.

Viewing pathological processes in this light we see that in general they represent either failures or distortions of one or more of the normal body mechanisms, in other words, the body does not invent new cellular mechanisms when confronted by disease or accident.

The reactions of bony tissue form no exception to this general rule. We see in diseased bones, firstly, pathological changes which are common to all tissues—namely (a) inflammatory reactions involving vessels and soft tissues inside or on the surfaces of the bones, (b) changes due to partial or complete vascular obstruction, e.g. necrosis of soft tissues and/or bone, and (c) tumours.

Secondly, we see reactions which are to a large extent peculiar to bone and which represent either failures or distortions of one or more of the normal processes of bone formation or bone absorption. The number of possible reactions of bone tissue is therefore strictly limited, and the wide range of histological change seen in bone diseases is accounted for by various combinations of the relatively few fundamental reactions.

In the following pages some of the more important of these fundamental reactions are discussed. No attempt is made to deal exhaustively with the pathology of individual diseases of bone—these are considered only in so far as they illustrate the points under discussion.

## I TYPES OF BONE STRUCTURE

Bony tissue consists of a collagenous matrix composed of fibrils embedded in a homogeneous substance ("cement"), the whole being rendered rigid by the deposition of calcium salts in a very finely divided state. This rigid matrix is honeycombed by numerous very fine canaliculi containing cytoplasmic prolongations of the bone cells which lie in small cell spaces scattered fairly uniformly throughout the bone. The fibrils of the bone matrix are collected into bundles forming coarser or finer fibres and the microscopic appearance of bony tissue is dependent on the size and arrangement of the fibres, to which is also related the arrangement and characters of the bone cells. The cell-

spaces and the canaliculi. Bone can be divided into two main types (a) *Lamellar* and (b) *Non lamellar* or *woven* bone.

**Lamellar Bone**—Lamellar bone is characterised histologically by the fact that its fibrils are arranged in sheets in each sheet forming a single lamella the general direction of the fibrils is the same but on passing to the next sheet of fibrils the general direction changes (as in plywood) as a rule through an angle of about 45 degrees though sometimes through a complete right angle. This alternation in the fibril direction produces the lamellar appearance which is most distinct where the change of angle is greatest. The regular arrangement of fibrils is associated with a regularity in the arrangement of the bone cells and cell spaces which tend to be elongated in the direction of the fibril. The canaliculi are long and well developed extending over the width of several lamellæ and anastomosing with those from neighbouring cell spaces (Figs 244 and 245).

**Non lamellar or Woven Bone**—Woven bone is characterised by the fact that the fibrils of the matrix are collected into bundles forming fibres of very varying diameter which cross one another in different directions forming an irregular woven felt work. This type of bone is usually deposited in the form of irregular coarse trabeculæ and when first formed shows ill defined brush like margins owing to the fact that many of the fibres emerge from the general felt work more or less at right angles and pass out to mingle with the surrounding connective tissue (Fig 252). The bone cells are more numerous and more irregularly distributed than in lamellar bone they are also larger and more irregular in shape (Fig 251). The canaliculi are short and poorly developed with few anastomoses.

Woven bone though usually formed as is lamellar bone by the activity of specialised connective tissue cells (osteoblasts) may occasionally be formed by metaplasia of already existing connective tissue. This is brought about by a deposition of a homogeneous ground substance (*cement substance*) between the connective tissue fibres the original connective tissue cell becoming enclosed in cell spaces to form the bone cells. The fibre system is in this case therefore that of the original connective tissue and in the matrix (fibres and ground substance) thus formed calcium salts are deposited. Such *metaplastic bone* is commonly found in the bony insertions of tendons and fasciæ. In some cases the interlacement of the fibres may be very slight e.g. in a tendon insertion they are nearly parallel.

#### *Note on the Nomenclature of Non lamellar Bone*

In the case of non lamellar bone there is no standard nomenclature in the English literature. *Koelliker* used the term *Faserknochen* or fibre bone some writers use the term coarse fibred bone. *von Ebner* (1850) used the term *geflechtartig* which is now in general use in the German literature and *H. M. Turnbull* has for many years used the equivalent English term woven. The last term seems the most suitable as it emphasises the woven felt work of fibres which characterises the matrix of this type of bone and distinguishes it from lamellar bone.

## II. OCCURRENCE OF THESE TWO MAIN TYPES OF BONE IN THE HUMAN SKELETON

**Lamellar Bone.**—Practically the whole of the post-infantile skeleton is made up of lamellar bone which forms two main types of gross structure: (a) compact bone (compacta), and (b) cancellous bone (spongiosa). The finer structure is essentially the same in both these types, and consists of groups of lamellæ, each group being demarcated from neighbouring groups by a "cement line" (q.v.) (Fig. 255a). The arrangement of the groups of lamellæ (lamellar systems) differs, however, in the two cases, and is determined largely by the blood-supply.

In the case of cancellous bone the trabeculae are for the most part thin enough to allow the bony tissue to obtain its nutrition from the surrounding blood-vessels in the marrow spaces. Compact bone, on the other hand, forms masses of bone tissue too thick to obtain their nutrition only from surface vessels, and is therefore perforated by special vascular channels, Haversian canals, around which the lamellæ form a series of concentric circles, each canal with its surrounding concentric lamellæ forming a Haversian system. Some of the larger trabeculae of cancellous bone also need such vessels, and may be seen to contain small Haversian systems. The numerous Haversian systems so characteristic of the compact bone of larger mammals and reptiles are therefore related to the mechanism for supplying blood to a considerable mass of compact bone; in small mammals and reptiles they are few or absent.

Non-lamellar, or Woven Bone forms the bony skeleton of the embryo, where it was first studied by *Gegenbaur* (1867); it is, however, gradually replaced by lamellar bone, so that after the first year of post-natal life practically the whole of the skeleton is normally composed of lamellar bone.

In many pathological states, however, woven bone is common: in fact it may be said that any bone tissue formed rapidly under pathological conditions will be of this type. Such pathological woven bone is, however, like the foetal woven bone, eventually replaced by lamellar bone: e.g. replacement of the woven bone forming the "provisional callus" of a fracture by the lamellar bone of the "definitive" callus.

### *Note on Terminology*

The removal of bone by osteoclasts, with the production of Howship's lacunæ, is often called by English writers "lacunar resorption." The term "resorption" is borrowed from the German expression; its English equivalent is "absorption." The term "apposition," used to express the laying down of bone, is also borrowed from the German, its English equivalent being "deposition." Throughout this article I have, except where quoting other writers, used the English terms "absorption" and "deposition."

## III. DEPOSITION OF BONE

It is important to realise that bone formation consists of two distinct processes: (1) the formation of the collagenous matrix, and (2) the calcification of this matrix.



**Formation of the Matrix**—In all types of bone other than metaplastic the bone matrix (collagen fibrils + homogeneous ground substance) is produced by the activity of specialised connective tissue cells (osteoblasts) some of which become incorporated in the cell spaces of the matrix as this is formed. These form the bone cells and thereafter appear to play no further part in bone formation.

One of the important distinctions between woven and lamellar bone lies in the fact that woven bone can arise in the middle of connective tissue while lamellar bone can only be laid down on an already existing surface of bone or calcified cartilage. Another difference is in the rate of formation. Lamellar bone the more complex type is deposited relatively slowly, woven bone the simpler type can be formed rapidly and can extend fairly rapidly over a region not previously occupied by bone or cartilage. In both lamellar and woven bone all the structural elements are formed before the calcium salts are deposited, even the canaliculi can be distinguished by suitable methods in this non calcified *osteoid tissue* (*Petersen 1930*). Under normal conditions calcification takes place very soon after the collagenous (fibres and ground substance) and cellular elements are completed. The deposition of calcium salts may to some extent obscure the structural elements but it does not change their arrangement in any way. In other words osteoid tissue has the same collagenous structure as the fully formed bone and cannot be distinguished from bone in completely decalcified specimens.

**The Calcification of Bone Matrix**—The first worker to make a thorough study of the histological features of calcification of the bone matrix was *Gustav Pommer (1885)*. He evolved a method of partial decalcification which allowed sections to be cut without destroying the hæmatoxylin staining reaction for calcium. By this method the calcified bone stains blue with hæmatoxylin while the non calcified osteoid tissue can be stained pink with eosin or some other suitable counterstain.

If *lamellar bone* is studied in sections treated by *Pommer's* technique it is found that the active bone formation is shown by the appearance of a narrow zone or seam of not yet calcified pink staining osteoid tissue on the surface of the calcified (blue staining) bone (*Fig 249*). A further indication of bone growth is seen in the evidence of increased activity of the osteoblastic cells on the surface of the osteoid seam. These cells are derived from the cells of the fibrous reticulum of the marrow or from those of the deepest layer of the periosteum. In the resting state they show flattened nuclei and scanty cytoplasm giving rather the appearance of a thin endothelium covering the bone surface. In the active state the cells increase in size and their nuclei are plumper and appear more crowded together (*Hunter and Turnbull 1931 p 255*). The calcification of the osteoid tissue which as mentioned above normally takes place soon after it is laid down is first seen as a deposit of fine hæmatoxyphil granules. These granules merge into a diffusely blue staining mass as the

process advances. Rapid growth is therefore shown by an active condition of the endosteal or periosteal cells at the bone surface together with a well developed osteoid seam. Lamellar bone in a resting (inactive) state shows no osteoid seam and its surface is covered by a single layer of spindle cells with scanty cytoplasm and sparsely scattered flattened nuclei. The process of calcification in woven bone is essentially the same as that in lamellar bone. Since however woven bone often shows an irregular margin the newly formed osteoid tissue forms an irregular peripheral zone and not a uniform seam as in lamellar bone (Fig 252).

A large amount of work has been done on the chemical mechanism by which calcium salts are deposited in the bone matrix particularly by *Robison* and his co workers. It appears however that further work will be required before the matter is completely elucidated and it is not proposed to discuss this question here. Bone matrix has a great affinity for calcium salts and except in rickets osteomalacia or allied conditions always calcifies soon after it has formed. There is no evidence that the chemical mechanism of calcification is reversible in the body. The removal of calcium from the skeleton is carried out by a different method—namely osteoclastic erosion. This question will be discussed more fully later.

#### IV THE NORMAL GROWTH AND RECONSTRUCTION OF THE SKELETON BY THE PROCESSES OF BONE EROSION AND DEPOSITION

During the process of skeletal growth it is necessary that great alterations in size and shape of the bones should be brought about without any interference with their supporting functions. The mechanism by which this is made possible could be demonstrated only after the development of a detailed knowledge of their minute structure.

*Tomes* and *de Morgan* (1853) were the pioneer workers in this field and considering the limitations of their technical methods their insight into this problem is surprising. They noted that bone and also the roots of milk teeth undergoing absorption presented finely pitted surfaces. In bone undergoing absorption under pathological conditions they observed that the Haversian canals were enlarged and irregular in shape. The same observation had already been made by *Hewship* (1819) but *Tomes* and *de Morgan* carried the matter farther and showed that even in normal bones spaces were produced by a similar process of erosion. Such spaces were often later filled up by the deposition on their inner surfaces of successive concentric lamellæ of bone with the final production of a complete lamellar or Haversian system. The mechanism by which Haversian systems were formed was thus demonstrated and *Tomes* and *de Morgan* named the eroded spaces Haversian spaces.

From such evidence as the above and from the evidence of the composite

"mosaic" structure of normal bone (*qv*) these authors concluded that the processes of absorption and deposition continue throughout life though less actively in older than in younger subjects. This view was subsequently confirmed by *Koelliker* (1873) *von Ebner* (1875) *Pommer* (1885) and other workers.

## V THE CEMENT-LINES AND THE " MOSAIC " STRUCTURE OF BONE

At the time when *Tomes* and *de Morgan* wrote their " Observations on the Structure and Development of Bone " it was known that a transverse section of the cortex of a long bone showed in addition to complete Haversian systems many partial and fragmentary systems known as interstitial lamellæ. Each of these fragmentary systems shows a group of lamellæ running in the same general direction and often forming a series of curves obviously corresponding in arrangement to fragments of a Haversian system. The general direction of the lamellæ in the various fragments is very different and each fragment is outlined and separated from its neighbours by a sharply defined line which is bitten out into a series of irregular shallow bays. The complete Haversian systems show more or less circular or oval outlines which are however, scalloped to fit into the bays in the surrounding bone. That is to say the space in the surrounding bone into which the Haversian system fits is always bitten out on its inside and in fact resembles exactly in shape a Haversian space formed by a process of erosion.

*Tomes* and *de Morgan* were the first to give a satisfactory explanation of this peculiar jig saw like arrangement of complete and fragmentary lamellar systems. It was clear that if each Haversian system was formed by the deposition of successive lamellæ on the inside of an eroded Haversian space such outlines would be produced. The irregular fragmentary systems could again be explained only as remains of older systems left over after previous erosion.

The whole question was again studied and completed in great detail by *von Ebner* (1875) who confirmed the general conclusions of *Tomes* and *de Morgan*. *Von Ebner* showed that the fibres of the bone matrix are interrupted at the irregular margins of the complete or fragmentary lamellar systems which are joined together by the non fibrous ground substance (' cement substance ' ) only, he therefore coined the term cement lines ( *Kittlinien* ). These cement lines appear as clear margins in ground-sections, in sections of decalcified bone well stained with hæmatoxylin they can be seen as fine irregular blue lines. *Von Ebner* showed further that the bone splits rather more readily along them than elsewhere, so that when broken apart at a cement line it shows two mutually fitting surfaces, a " mould " lined with pits (*lacunæ*) and a " cast ". The cast shows a series of small bosses fitting into the pits in the mould. The surface of the mould shows the characters

of an eroded bone surface its lamellar systems and canaliculi being cut off sharply, as with a knife. In the cast the lamellæ run nearly parallel to the surface, and the canaliculi are not cut off but turn back in a series of loops, giving the appearance of a natural boundary (Figs 245a and 245b). This corresponds exactly to what is seen in a freshly deposited layer of lamellar bone covering an eroded surface. The cement lines therefore correspond to old surfaces of erosion covered with newer bone and thus incorporated in the bony structure.

This mosaic structure, although most obvious in sections of the compact bone of the cortex of a long bone, is present to an equal degree in the trabeculae of cancellous bone.

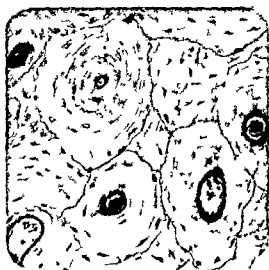


FIG. 244.—Transverse section cortex femur showing Haversian systems composed of concentric lamellæ also fragmentary ('interstitial') lamellar systems. The various systems are separated from one another by irregular cement lines.  $\times 130$ .



(a)



(b)

FIG. 245(a)—Semi-diagrammatic picture of parts of two Haversian systems separated by a cement line. The larger Haversian system on the right has been partly eroded and the smaller system has been laid down in the space so produced. The cell-spaces and canaliculi are shown as stained by Schmorl's thionin blue method.

(b)—Shows the two Haversian systems seen in Fig. 2(a) separated along the 'cement line'. The eroded space in the larger system on the right forms a mould into which the smaller system fits as a cast (vide text Section 1).

The whole skeleton although formed of bone which is mechanically a continuous structure is in fact composed in the form of a jig saw the individual pieces of which were deposited at different times during the life of the individual (Fig 244)



FIG 246.—Bone in Paget's osteitis deformans showing a jig saw like pattern produced by numerous cement lines the result of repeated alterations of bone erosion and deposition. 100

one looks at a transverse section of the cortex of a long bone suitably stained with hæmatoxylin one sees in places particularly in the zones of ensheathing lamellæ beneath the periosteum and in the central lamellæ lining the medullary cavity bluish lines separating groups of lamellæ. These lines differ from the cement lines of erosion in that they show a smooth and not a bitten out outline and that the canaliculi do not show a bitten off appearance (Fig 247)

A similar line of arrested deposition forms on the bone surface surrounding the Haversian canal in the completed Haversian systems. It has been shown that such lines form when a bone surface remains for some time in a resting state without the occurrence of either deposition or erosion. Petersen (1930 Fig 31 in his work)

In young children in whom the processes of erosion and deposition have extended over a shorter period one would expect a less fragmentary structure this is in fact what is found (von Ebner 1875). In diseases where there has been a great deal of bone erosion with new bone formation (e.g. Paget's disease of bone) the mosaic structure becomes much more marked i.e. the jig saw is composed of smaller pieces (Fig 246) (Schmorl 1930 and Hunter and Turnbull 1931)

#### Lines of Arrested Deposition—If

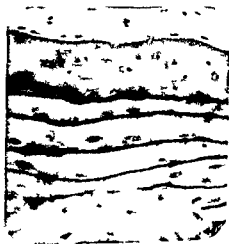


FIG 247.—Lines of arrested growth separating groups of circumferential lamellæ beneath the periosteal surface of the femur. The lamellæ themselves are not visible. The lines show smooth contours, with the exception of the uppermost which is not a line of arrested growth but a cement line at the site of a previously eroded surface. A comparatively wide zone of newly formed bone lies superficial to this line.  $\times 200$

shows a section of a stag's limb bone with a well developed circumferential line of this type produced by an arrest of growth during the winter. In the earliest stages of bone erosion on the inside of a Haversian canal a striking picture is produced by the eating away of this line of arrested deposition over part of its circumference (Fig. 259)

## VI THE PROCESS OF BONE EROSION—OSTEOCLASTS

*Tomes and de Morgan* (1853) although their technical methods evidently did not allow them to distinguish easily different types of cell considered that the erosion of bone came about by the activities of the masses of cells of the soft tissues which filled the erosion pits (later called Howship's lacunæ) on the surfaces of bones and milk teeth undergoing absorption.

Following on this work various observers saw and described large multinucleated cells on bone surface.

*Koelliker* (1873) made more extensive studies of these multinucleated cells and their role in bone absorption and coined the term *Ostoclast* later converted into *Osteoklast* or *Osteoclast*.

**Characters of the Osteoclast**—The fully developed osteoclast is a large multinucleated cell varying in size from about 45 to 90  $\mu$  in its longest diameter. Its large size and its numerous rounded nuclei make it easy to distinguish from the various other cells in bone surfaces and in marrow spaces (Fig. 248).

In addition to the fully developed multinucleated osteoclast one finds all grades of smaller varieties. The smaller cells contain 2–5 nuclei (Fig. 259) while very large specimens may contain dozens.

Again gradations are seen between the smaller osteoclasts and the cells of the fibrous reticulum (spindle-cell connective tissue so called endosteum) which separates the inner surfaces of the bone from the blood forming or fatty marrow.

As to the origin of the osteoclasts there can be little doubt that they can arise from any of the spindle cells of the fibrous tissue clothing the bone surfaces (a tissue from which the osteoblasts are also derived). In other words, these functionally inactive spindle cells can with a suitable



FIG. 248.—Active osteoclast. The bone which appears black in the illustration is undergoing erosion. Two large multinucleated osteoclasts are seen lying in Howship's lacunæ (near upper and lower margins of illustration). There are also other smaller osteoclasts and a fine reticulum of endosteal connective tissue occupies the marrow space. / 340

stimulus at any time undergo a further development into either of two histological types whose characters are correlated with the activities of either bone formation or bone destruction. Similar potentialities are found in the connective tissue cells of other parts of the body but the necessary stimulus for bone formation is only exceptionally produced in parts distant from the bones such a stimulus may be furnished by the introduction of fragments of dead bone and it may also be produced near a mass of calcified material—e.g. a calcified tuberculous focus.

As regards the formation of cells of the osteoclast type from the ordinary connective tissue this always occurs if a non infected foreign body is introduced into the tissues. This results in the production of multinucleated foreign body giant cells which show histological characters indistinguishable from those of the osteoclast. These cells apply themselves to the surface of the foreign body which thereby undergoes gradual dissolution. If fragments of clean boiled bone are introduced into the tissues the foreign body giant cells erode them and behave as far as can be judged exactly as osteoclasts. Large multinucleated cells of this type are obviously adapted functionally for the removal of solid fragments which are too large to be completely engulfed. The large mobile protoplasmic cell body can be applied to a surface and can exert a solvent action at the surface of contact. When a solid body is reduced to sufficiently small fragments these become completely engulfed by such cells and are then digested intracellularly. A foreign body of moderate size e.g. a piece of sterile catgut is often completely ensheathed by a collection of multinucleated cells. In the case of the much more extensive bone surfaces however the osteoclasts are not sufficiently numerous to form a continuous sheet they occur in groups or as isolated cells. The digestive action of one of these cells on the bone surface results in the formation of a small pit—usually shallow in which the cell comes to lie thus producing the erosion pits (or Howship's lacunæ) the process of removal of bone by this means being therefore called *lacunar absorption* (or resorption). A study of the edges of erosion pits in sections prepared so that the calcified and non-calcified matrix can be distinguished shows that in the process of osteoclasts the whole of the bone tissue both calcium and organic matrix is removed leaving a perfectly sharp edge as if the tissue had been taken out by a punch. There is no sign at all of decalcification of the bone matrix on the edge of the erosion pit nor is there any sign of injury to the bone cells in the neighbouring lacunæ. Although one must suppose that proteolytic and possibly decalcifying substances are secreted by the cell at its surface of contact with the bone there is no evidence that the action of these substances extends beyond this surface. This can be seen in Fig. 249 which shows a trabecula of bone undergoing lacunar absorption on its lower surface.

Osteoclasts therefore erode living bone by a process (which might be called *chemical chiselling*) which causes no visible alteration or injury to the cells

or matrix even on the very edge of the line of erosion. Since osteoclasts is the normal process by which the internal structure of the bones is remodelled during growth, it is obviously necessary that there should be no weakening of the matrix which remains on the edge of the eroded zone. The mechanism just described, by which the erosive effect is limited to the surface of contact of the osteoclast with the bone, seems admirably adapted to this end. Osteoclasts appear to erode living and dead bone with equal ease, and it is of course well known that osteoblasts can deposit new bone on a surface of dead bone.

**Fate of the Osteoclasts**—The fully developed osteoclasts can probably survive for only a short time, but their exact fate has never been precisely determined. Degenerative changes can be seen in them, and it seems probable that they disintegrate locally. Calcium salts must escape from the osteoclasts either by disintegration of the cells or by discharge of the products of their activities, for it is quite clear that the calcium derived from the bone which they have removed finds its way eventually into the blood stream (Subsect xx). The microscopic appearances of bone removal by osteoclasts and the evidences of very extensive past osteoclasts throughout the skeleton can be observed by anyone who can make good sections of bone, and the evidence is to the functional activity of these cells has been generally accepted by all serious workers on the histopathology of bone since *Koelliker's* time. Nevertheless, assertions are made from time to time that these cells play either (1) no part in the removal of bone, or (2) only a minor part.

There can be no doubt as to the bone-eroding powers of osteoclasts, the second question, whether they play a major or a minor part in bone destruction and the removal of calcium from the skeleton will be discussed later.

One histological point that has sometimes given rise to doubts as to whether the erosion pits are produced by osteoclasts is the fact that one often sees specimens in which only a few of the erosion pits contain these cells.



FIG. 249.—A small trabecula of bone surrounded by a sheath of endosteal connective tissue beyond which is fatty marrow. Section prepared to show the distribution of calcium which is stained dark while a seam of newly formed non-calcified osteoid tissue running along the upper border of the trabecula appears light. This seam is covered by a row of osteoblasts. The lower edge of the trabecula is undergoing osteoclastic absorption and two large dark staining osteoclasts are seen separated from their lacunae on the bone surface by contraction of the tissue during preparation. These lacunae show clean-cut outlines and there is no loss of calcium staining at the eroded surfaces.  $\times 260$ .



A little consideration will however show that this is only to be expected. The osteoclast has the characters of an actively phagocytic cell and as we have mentioned above it appears to have a relatively short life. The pits on the bone surfaces however remain indefinitely until they are either covered by the deposition of new bone (in which case they remain as a cement line of erosion) or until the bone is further eroded with the production of another set of pits. It follows therefore that only those pits in which active erosion was going on at the time of removal of the bone specimen will show osteoclasts *in situ*.

### VII CAN THE REMOVAL OF BONE WITH RESULTING POROSIS BE BROUGHT ABOUT BY PROCESSES OTHER THAN CELLULAR EROSION BY OSTEOCLASTS?

The removal of bony tissue can be brought about by an active cellular erosion which removes both calcium salts and bone matrix together leaving a sharply defined punched out margin and causing no decalcification or other defect in the quality or structure of the bone which remains behind. With this view all serious workers on the histopathology of bone will agree.

The question whether there are other processes by which either (1) the entire bone (matrix + calcium) or (2) the calcium alone can be removed will now be discussed.

**Can Cells other than Osteoclasts remove Bone?**—It has been suggested that cells other than fully developed osteoclasts are capable of eroding bone particularly (a) fibroblastic cells of granulation tissue which have not developed the recognisable histological characters of osteoclasts and (b) tumour cells.

As regards (a) the fibroblastic cells we have seen that these give rise to osteoclasts and that smaller binucleated types of osteoclasts are often found in the erosion pits (Howship's lacunæ). It is obviously not possible to say at what exact stage of development the osteoclastic properties appear but it is clear that areas in which active erosion is taking place always show differentiated osteoclasts. (b) It is well known that tumour cells invade bone and lead to porosis but as will be seen later evidence points to the fact that this erosion is produced by stimulation of osteoclasts and not by a direct erosive action of the tumour cells.

**Are there processes due not to erosion by living cells but to some sort of chemical break down of the bone by which it reverts to a non calcified fibrous matrix?**—Processes by which the calcium can be absorbed from bone with or without a subsequent break down of the decalcified collagenous matrix have been described by various writers. The earliest of these processes was *haliteresis* a term meaning salt deprivation used by Virchow to indicate a hypothetical process by which the calcium salts were removed from the bone matrix in rickets and osteomalacia. More recently Pellerer (1906) and Leriche and Policard (1926) have described a rather similar process.

which the latter writers call 'osteolysis' In this process in addition to removal of calcium, the bone matrix is said to revert to fibrous tissue by a process of "breakdown" or 'de-differentiation' There is no satisfactory histological evidence for the existence of any of these processes, nor is it necessary to assume their existence in order to explain the appearances seen in normal and pathological bones This question is discussed below

### VIII THE FORMATION OF WOVEN BONE UNDER PATHOLOGICAL CONDITIONS

One of the difficulties in the interpretation of the histological appearances of diseased bones arises from failure to distinguish between the two main types of bony tissue, namely (1) lamellar and (2) woven bone A general description of these two types has already been given and it has been pointed out that lamellar bone can be formed only on an already existing surface of bone or calcified cartilage while woven bone may arise either on an existing bone surface or in the middle of connective tissue mass The formation of trabeculae of woven bone in a connective tissue mass is very common in pathological conditions Inflammatory or other changes which produce fibroblastic proliferation in soft tissues when they occur in relation to bone, result in a similar proliferation of endosteal or periosteal fibroblasts which is followed by the formation of irregular trabeculae of woven bone in the connective tissue mass so produced In this way a considerable mass of new bone can be formed rapidly, e.g. the provisional callus round a fracture and the new bone in subacute periostitis This rapidly formed woven bone is eventually either removed by osteoclasts or forms a scaffolding on which lamellar bone is deposited In either case if the patient survives long enough the remodelling process finally results in the entire removal of the woven bone only lamellar bone remaining

Mixtures of woven and lamellar bone may present confusing pictures, some of which appear to have been the basis for the erroneous belief that bone can 'break down' and revert to a fibrous matrix

The following combinations of the two types may occur

**Woven Bone forming on a Surface of Lamellar Bone**—This often occurs in inflammatory conditions In most cases the surface of the lamellar bone has undergone some osteoclastic erosion and is pitted by Howship's lacunae On this irregular surface a layer of woven bone forms and often extends into the surrounding connective tissue as irregular trabeculae (Figs 250, 251) In some areas the newly formed woven bone usually shows a brush like margin, with the main fibre direction at right angles to the lamellar bone surface In sections prepared to show the distribution of calcium this brush like margin is seen to be still uncalcified (Fig 252)

**More Intimate Mixtures of Lamellar and Woven Bone**—Bone trabeculae may be composed partly of lamellar and partly of woven bone In normal



FIG 250 Irregular trabeculae of woven bone extending to the surface of the thickened periosteum of a long bone in a case of subacute periostitis  $\times 5$



FIG 251.—Portion of Fig 250 showing irregular trabeculae near surface. The numerous large bone cells in irregular closely packed bone spaces are characteristic of woven bone  $\times 100$



FIG 252 Calcifying woven bone. In the upper part of the figure (A) a non-calcified fibrous edge of woven bone mingles with the surrounding connective tissue. The calcifying more darkly stained zone (B) shows at its edge fine dark staining granules of calcium. This woven bone has been in part eroded (*vide* irregular cement line separating zone B from zone C) and on it has been deposited fresh bone which at C is lamellar in structure (lamellae not visible here). Below the granular calcifying edge at C is a seam of lamellar osteoid tissue (D)  $\times 250$

bone the trabeculae show a mosaic appearance indicating that they are composed of fragments of different ages. These fragments are ordinarily all composed of lamellar bone but in the foetus and young child and under pathological conditions a trabecula may be made up in part by lamellar and in part by woven bone the arrangement depending upon the sequence of changes which has occurred. Thus if a trabecula of lamellar bone has been reduced by osteoclastic erosion it may have subsequently been added to by the rapid formation of new woven bone on the remains of the old bone so that a trabecula of woven bone containing a core of eroded lamellar fragments is produced. The reverse process takes place during the remodelling of a mass of rapidly formed woven bone, such as occurs during the process of replacement of a provisional by a definite

tive " callus Here the whole of the woven bone is eventually removed by osteoclastic erosion but the process is a very gradual one In the early stages many of the trabeculae of woven bones serve as scaffoldings on which lamellar bone is deposited These scaffoldings may be either intact woven bone trabeculae or remnants of such trabeculae reduced by osteoclasia In the case of intact trabeculae which have been further added to by the



FIG 253—Part of a trabecula composed of a core of woven bone on which a shell of lamellar bone has been deposited The woven bone has undergone extensive osteoclastic erosion as can be seen from the broken out margins of the cement line of erosion which bounds it Two large irregular bays have been eroded these are now lined by layers of lamellar bone and converted into vascular spaces with smooth walls  $\times 200$



FIG 254—The same section as in Fig 253 photographed by polarised light to show the fibre structure of the bone The lamellar structure of the ensheathing bone is well seen on the right hand side and can be distinguished from the irregular feltwork of the woven bone forming the core  $\times 200$

deposition of surface layers of lamellar bone, there is a gradual transition from a core of woven bone to a lamellar arrangement on the surface Lamellar bone may be deposited directly on the eroded surface of a trabecula of woven bone in this case the resulting trabecula will show the two types of bone separated by a cement line of erosion—an abrupt transition (Figs 253, 254) The gradual replacement of a mass of woven bone by lamellar bone involves much alternating erosion and deposition of both woven and lamellar bone the final form being largely determined by the strains and stresses to which the tissue is subjected There is no evidence that the final disappearance of the woven bone is due to its special susceptibility to osteoclastic erosion Osteoclasia of both woven and lamellar bone takes place freely for some time, but after the more active stage has passed

all the new bone formed is of the lamellar type, and finally none of the original woven bone remains.

#### IX. HISTOLOGICAL EVIDENCE AGAINST THE EXISTENCE OF A PROCESS BY WHICH BONE "DE-DIFFERENTIATES" INTO CONNECTIVE TISSUE

It is of course clear that in various pathological processes where bone undergoes osteoclastic erosion, its place may be taken by spindle-cell connective tissue of either periosteal or endosteal origin. Such a process takes place extensively in generalised osteitis fibrosa. As mentioned above, however, certain writers believe that bone can disappear without the action of osteoclasts by a process of "breaking down" or "de-differentiation" into connective tissue. *Leriche* and *Policard* (1928) describe such a process under the term "osteolysis." Since their views on this matter have been accepted by certain authorities in this country without criticism, it seems advisable to point out why it is impossible to accept them, on histological grounds.

Firstly, the description of the histology of this process is most unconvincing, and it is clear that these workers are mistaking bone formation for bone absorption, (the authors themselves admit that they cannot distinguish these two processes, for they say at the end of their description: "It is, moreover, impossible to distinguish these histological forms of resorption by osteolysis from those of the formation of bone"). This mistake has been made before by other workers, e.g. *Retterer* (1906, Fig. 6, p. 219 of his work), who illustrates the reversion of bone to fibrous tissue by a figure which shows typical woven bone *forming* on the surface of more compact (probably lamellar) bone. This criticism of *Leriche* and *Policard's* views has already been made by *Weidenreich* (1930).

Secondly, careful histological studies using methods which display the fibre systems of the bone matrix afford strong evidence against any form of "breakdown" of bone into connective tissue. In the case of lamellar bone which, as previously remarked, forms almost the whole of the normal skeleton, the difficulties of assuming such a "breakdown" into connective tissue are even greater than with woven bone. As already noted, lamellar bone has a very complex fibre system, different from that of woven bone and quite different from that of ordinary connective tissue. It could not therefore arrive at anything resembling ordinary connective tissue by a process of disintegration, but would have to be picked to pieces, sorted into bundles, and put together again in a different pattern. A form of "breakdown" which would do this is inconceivable. In fact, lamellar bone retains its characteristic structure even when reduced to minute fragments by the action of osteoclasts.

In their discussion of osteolysis *Leriche* and *Policard* state that the process is seen in its clearest form in cases of generalised osteitis fibrosa. Since 1926, when their book appeared, there has been a considerable increase in our

knowledge of generalised fibrocystic disease and its relationship to hyperparathyroidism and further careful studies have been made by *Hunter* and *Turnbull* (1931) and others. These investigations lend no support to *Leriche* and *Policard's* belief in the existence of osteolysis, but on the other hand stress the great importance of increased osteoclastic absorption in this disease.

## X CHANGES IN THE CALCIUM CONTENT OF BONE

The general use of the term "decalcification" for all conditions in which the X-ray density of bones is reduced has led to much confusion of thought. When the X-ray density of a bone is reduced we know that the bone as a whole contains less calcium salts than normal, but this reduction in the amount of calcium may be due to two distinct conditions.

(1) *Osteoporosis*—Here there is a rarefaction of bone brought about by a removal of bone substance by osteoclasts. The trabeculae are thin, and the Haversian canals of the cortex are enlarged to Haversian spaces which microscopically show irregular margins covered with Howship's lacunae, many of which are occupied by osteoclasts. The total bony tissue is thus much reduced in amount, what remains, however, shows no histological evidence of loss of calcium, i.e. there is no increase in the amount of osteoid tissue, and on chemical analysis the individual fragments have a normal calcium content (*S. L. Baker, W. W. Kay, 1937*).

(2) *Hypocalcification*—The writer uses the term "hypocalcification" to describe the condition met with in rickets and osteomalacia, diseases in which the bony tissue contains less calcium than normal. This is shown histologically by the fact that there is an abnormal amount of non calcified osteoid tissue, and chemically by the fact that an analysis of individual fragments shows an abnormally low calcium content. The term "hypocalcification" is, I think, preferable to the term "decalcification," because it expresses the fact that the bone is not fully calcified without assuming that the calcium has been abstracted, as does the expression "decalcification." As shown below, hypocalcification is not produced by abstraction of calcium (halisteresis), but by a failure to calcify. Fig. 255 shows diagrammatically the conditions in normal bone (*a* and *b*), porosis (*c*), and hypocalcification (*d*). It is clear that in either (*c*) or (*d*) the X-ray density would be decreased.

It might be thought that the surgeon could tell by cutting through the bone whether there was excess of osteoid tissue or not, and bones are often described as being soft and "decalcified" because they can be easily cut into bones which are merely porotic will, however, cut easily owing to the hollowing out of the compact cortex and the fineness of the trabeculae of the spongiosa.

## XI ESSENTIAL BONE CHANGES IN RICKETS AND OSTEOMALACIA

*Pommer* (1885) was the first worker to make a thorough histological study of these diseases, using methods which would distinguish the calcified bone

from the non calcified osteoid tissue Before *Pommer's* work the whole subject was chaotic, there was no agreement as to whether these two conditions were essentially the same, and it was believed by many that the origin of these diseases lay in some defect in the skeleton itself *Pommer* threw an entirely new light on the matter, and although vitamins were of course unknown at that date, he foreshadowed the discovery of this factor in a remarkable way

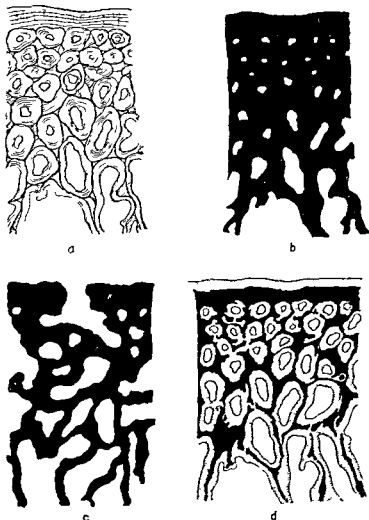


FIG 255.—Diagrams of transverse sections of cortex of long bones, showing the reduction in calcium content of the skeleton resulting from (1) porosis and (2) hypocalcification. (a) Normal lamellar structure, showing from above downwards subperiosteal lamellae, Haversian systems, and trabeculae springing from wall of medullary cavity. Thin lines = lamellar boundaries, thick lines = "cement lines", (b) outline of the same, showing normal calcified bone (black), (c) porosis, bone fully calcified but deficient in amount, (d) hypocalcification as seen in long standing rickets or osteomalacia. A large amount of bone is non-calcified osteoid tissue (shaded), this would be invisible radiologically.

The final conclusion from his extensive studies of rickets and osteomalacia were

(1) The essential defect in both these diseases is the same, namely a defect in the calcification of any new bone formed during the disease

(2) There is no evidence of "halisteresis" i.e. there is no abstraction of calcium from such bone as was already calcified when the disease started

(3) The softening of the skeleton is explained by the fact that during the normal growth and reconstruction of the bones the old bone formed and calcified before the onset of the disease is gradually removed by osteoclastic erosion and replaced by new, if the new bone fails to calcify or calcifies imperfectly the skeleton becomes gradually softer the soft bone (osteoid) being naturally most abundant and obvious where new bone formation is greatest

(4) There is no evidence that this osteoclastic erosion is more rapid in rickety subjects than in normal children of the same age

(5) The disease is not due to any cause in the bones themselves, but to some factor outside the skeleton which interferes with the proper calcification of new bone formed during the disease

Pommer's views met with a considerable amount of opposition. The question of the occurrence of halisteresis was still being vigorously debated in 1909 (Verhand Deutsch Gesell., 1909 pp 54-67) and is considered by some writers to be still unsettled. More recent work on rickets and osteomalacia has however, certainly confirmed Pommer's views

## XII EVIDENCE AGAINST HALISTERESIS

The following is some of the evidence indicating that the bone changes in rickets and osteomalacia are due to failure of calcification of new bone and not to abstraction of calcium from already calcified bone matrix

If the changes in rickets and osteomalacia were produced by a process of abstraction of calcium from the bone matrix this would operate on the already calcified bone present at the beginning of the disease, if on the other hand, the process were purely a failure to calcify it would affect only the new bone formed during the disease

The fact that the development of non calcified tissue (osteoid) corresponds with the deposition of new bone as regards (1) site and (2) rate of growth shows that the process is a failure to calcify. The evidence is (a) clinical (b) microscopical and (c) chemical. The clinical evidence is obvious only in rickets

### Clinical Evidence

SITE.—The development of non-calcified tissue occurs rapidly at the growing epiphyses in rickety children. In the shafts of the bones, where the replacement of old bone by new is comparatively slow the softening process develops



slowly If new periosteal bone is formed this develops as a zone of soft tissue outside the old shaft

**RATE OF GROWTH OF BONE**—Florid rickets occurs in well nourished growing children when bone growth is slow (malnutrition or scurvy) rachitic changes are slight or absent

### Microscopic Evidence

**SITE**—The osteoid tissue is seen first and most abundantly in the zone of endochondral ossification at the epiphyses where there is also failure of provisional calcification of the cartilage Rachitic changes are also most marked at those epiphyses where growth is most rapid (*Maxwell Hu and Turnbull 1932*) In other situations in both rickets and osteomalacia the osteoid tissue develops where new bone is being formed on the trabecular surfaces and on the inside of Haversian systems In these situations narrow seams of osteoid tissue are normally present in rickets and osteomalacia the width of such seams is greatly increased

**RATE OF GROWTH**—The rate of growth can be roughly judged by the appearances of the osteoblasts Where osteoblastic activity is slight the rachitic or osteomalacic changes are least marked (*Hunter and Turnbull 1931 p 200*)

### Evidence from Metabolic Experiments

Metabolic studies have shown that the rate of removal of calcium from the skeleton is not increased in rickets it is increased in diseases in which there is increased osteoclastic activity (e.g. hyperparathyroidism) This question will be discussed more fully later when the conditions associated with increased osteoclasts have been considered

## XIII CONCLUSIONS ON THE MECHANISMS OF BONE DEPOSITION AND ABSORPTION

(a) Bone formation takes place in two stages

(1) The formation of the organic matrix of the bone by the action of specialised connective tissue cells viz osteoblasts

(2) This matrix is converted from the condition of non calcified osteoid tissue into true bone by the deposition of calcium salts throughout its substance

(b) Bone removal takes place in one stage and is carried out by multinucleated macrophages which remove the organic matrix and calcium salts together

(c) Calcium can be removed from the skeleton only by the action of these osteoclastic cells—the process being histologically not decalcification but 'de-ossification'

There is no evidence for the existence of such processes as "halisteresis" or the "osteolysis" of *Leriche* and *Policard*, which imply a dissolving out of calcium from the bone matrix or a "breaking down" or "de-differentiation" of the matrix into connective tissue.

(d) These conclusions apply not only to normal but to all pathological conditions of bone.

## CHAPTER XIX

### GENERAL PATHOLOGY OF BONE (*continued*)

#### BONE DEPOSITION AND ABSORPTION UNDER PATHOLOGICAL CONDITIONS

##### XIV MECHANICAL FACTORS

INCREASED STRESS or strain produced either by excessive use or by alterations in the mechanical conditions of the part is a well recognised cause of a local increase in bone production which is compensatory in nature. Any pathological condition resulting in a weakening and/or deformity of the bone tends to produce a compensatory increase in bone formation. The production of a mass of new periosteal bone (or osteoid tissue) tending to fill and buttress the concavity of a bent rachitic bone is a good example of this mechanism. The same buttressing effect is seen in the filling up of the concavity of an angular deformity in a malunited fracture. On the convexity of the malunion, owing to diminished mechanical stimuli, little or no deposition occurs but osteoclastic absorption continues. These factors tend in time to restore the line of the limb.

A large amount of work has been done on the reactions of bone to mechanical strains and stresses, and it may be taken as a well established fact that osteoblastic activity is in some way stimulated by increased tension or compression. The exact mechanism of such stimulation, however, remains obscure. It seems probable that bending movements in the bone structure with or without microscopic inflections furnish the necessary stimulus. In longstanding rickets or in osteomalacia the trabeculae of the spongiosa, e.g. of the ribs or vertebrae, are usually much thicker than normal owing to the increased deposition of lamellar osteoid tissue on their surfaces. As *Turnbull* notes (*Maxwell Hu and Turnbull*, 1932, p. 431), there is little doubt that this is the result of normal stresses on abnormally soft bones. This would support the view that the stress stimulus acts by producing some distortion of the bone structure with resulting stimulation of osteoblastic activity. In the case of normally calcified bones, bending effects would decrease with the increased rigidity resulting from the bony thickening and the stimulus to osteoblastic activity would thus rapidly diminish, in the case of rachitic or osteomalacic bones, however, depositions of soft osteoid tissue would not prevent bending the continued effects of which would stimulate excessive osteoblastic activity and result in thick osteoid deposits.

Absence of the normal mechanical stimuli appears to be the main factor in the production of the moderate grades of disuse atrophy which develop slowly. The mechanism in this case is the same as in senile bone atrophy, viz failure of osteoblastic activity, so that the normal osteoclastic erosion is not counterbalanced by a normal bone deposition.

As regards the more rapid acute types of bone atrophy, there can be little doubt that there is, in addition to poor deposition of bone, an increase in osteoclastic absorption. The view has been put forward that changes in the blood supply play an important part in this type of atrophy. This question will be considered later when discussing factors influencing osteoclasia.

## XV NECROSIS OF BONE

Necrosis of bone is characterised histologically by the loss of nuclei and disappearance of the bone cells, so that the cell spaces in the bone matrix appear empty (Fig 258). The bone matrix itself remains unchanged in appearance until removed by osteoclasts. The naked eye appearance of dead bone is due not to an alteration in the appearance of the osseous tissue itself but to absence of vascularity, dead bone which has been revascularised appears like living bone.

Dead bone shows no radiographic change until it has been demarcated from its surroundings by a line of separation. The statement has often been made that dead bone requires a greater radiographic density because of an increase in calcium content, and some writers appear to regard this supposed increase in calcium content as analogous to the calcification of a mass of dead tissue such as occurs for instance in a caseous focus. There is, in fact, no evidence that dead bone matrix acquires additional calcium. There are only two known ways in which the increased radiographic density of dead bone can be produced: (1) by contrast with a surrounding porosis, or (2) in the case of cancellous bone by crushing and compaction of the trabeculae into a solid mass of bone debris. A relative density is seen in sequestra, which may be equal in density to the original bone, while the surroundings have nearly always undergone a marked porosis. An increased density due to compaction of bone debris may be seen in such conditions as *Köhler's disease* of the tarsal scaphoid.

**Reaction of the Tissues to Dead Bone**—Non infected dead bone produces no appreciable inflammatory reaction when introduced into the tissues. There is a local proliferation of blood vessels and fibroblasts, and the development in the fibroblastic tissue of foreign body giant cells, which, as mentioned above (Sub sect vi) resemble multinucleated osteoclasts in appearance, and function as such by producing lacunar absorption of implanted bone. This newly formed tissue tends to penetrate all the spaces (cancellous and Haversian) in the bone and any necrosed soft tissue remaining in these spaces is removed.

by phagocytosis. The dead bone thus becomes revascularised and its spaces filled by a living connective tissue (Fig 257). Complete removal of the bone by osteoclastic erosion on both the internal and outer surfaces may follow this revascularisation. If however the invading fibroblastic tissue is osteogenic as it will be if the dead bone fragment has been inserted in or near living bone it will lay down new living bone on the partially eroded surfaces both internal and external of the dead bone. In this way a fragment of dead bone forms a scaffolding for and becomes incorporated in a mass of newly formed living bone where it may remain for months or years.

This peculiar acceptance of dead bone by the body and its incorporation in a newly formed bone structure accounts for the success of bone grafts as a method of repair. Although a bone graft may be living at the time of insertion the severance of its blood supply leads inevitably to the death of all but such superficial cells as can obtain sufficient nourishment from the surrounding tissue juices. These superficial osteoblastic cells together with those from any living bone in the neighbourhood proliferate and supply the osteogenic tissue which reclothes the graft with living bone. The revascularised portions of a graft appear like living bone to the naked eye and blend with the surrounding tissue. Microscopically the bone spaces contain living vascular tissue and they are lined by a layer of living bone. This zone of living bone is usually bounded by a cement line of erosion (*q.v.*) beyond which the bone is dead and shows empty cell spaces.

This acceptance of dead bone and its revascularisation with or without subsequent complete osteoclastic absorption is to be regarded as the normal reaction. The rejection of dead bone in the form of a sequestrum surrounded by pus is a complication due to an infective process. The development of such a sequestrum is considered below.

**Aseptic Bone Necrosis**—Interference with the blood supply is a potent cause of necrosis of bone. The blood supply may be interfered with in various conditions e.g. inflammations, vascular disease or trauma.

Obstruction of the blood supply to a portion of bone without infection produces an area of aseptic necrosis—an infarct of bone. The production of such aseptic necroses of bone has been extensively studied by *Axhausen* who has found them to be the basis for the puzzling group of localised bone lesions which includes such conditions as *Perthe's* disease of the hip, the disease of the head of the second metatarsal and that of the tarsal scaphoid described by *Köhler*. *Keinbock's* disease of the semilunar, *Schlatter's* disease of the tubercle of the tibia and the condition first described by *König* under the term 'osteochondritis dissecans'. All these diseases were described on the basis of X-ray and clinical findings and opportunities for studying their underlying pathology have been few. A detailed account of their histopathology will however be found in a contribution by *Axhausen* and *Bergman* (1937). It will be sufficient here to outline some of the more important

histological reactions underlying the peculiar series of changes observed radiographically in these conditions. A portion of bone deprived of its blood supply undergoes necrosis affecting both the bone cells and soft tissues within the bone. Since neither osteoclastic absorption nor bone formation can take place without a blood supply, such a mass of dead (infarcted) bone remains for a time architecturally intact, showing no loss of continuity with its surroundings or other changes that can affect the X ray picture. After a time, however, blood vessels and fibroblasts from the surrounding living tissue begin



FIG. 256.—Part of cancellous bone of the head of the femur near the articular surface (seen top left). There is complete necrosis of both bone and soft tissues, but the architecture remains intact except for a clear circle of rarefaction in the centre. This corresponds to the middle of a revascularised zone (darker area) in which bone absorption has taken place.  $\times 2$ .



FIG. 257.—Shows edge of reticular section in Fig. 256. The lower and right-hand part of the figure shows a darker staining necrotic tissue containing numerous vascular loops. This extends upwards into an area of isolated completely necrotic marrow tissue lying in the cancellous spaces between necrotic bone trabeculae. Mallory's connective tissue stain (modified).  $\times 80$ .

to invade the necrosed portion. Since the fibroblastic tissue is usually derived from the surrounding bone or periosteum it is capable of giving rise not only to osteoclastic but to osteoblastic cells, with the result that both absorption of dead bone and the deposition of new bone can take place in the revascularised zones. With this revascularisation of the infarcted bone X ray changes become apparent.

The rate of reorganisation of a mass of dead bone depends very much on its size and on the available blood supply. For instance, in the case of necrosis of the head of the femur following fracture of its neck, the blood supply available from the surroundings may be very limited. Fig. 256 shows a low power view of a portion of a necrosed femoral head undergoing

focal revascularisation by small vessels from the region of the ligamentum teres

A high power view of the advancing vessels is seen in Fig 257. The absence of nuclear staining of the bone cells and the marrow tissue shows that the whole of the head is necrotic. There is no indication of this necrosis in the low power view because the architecture as a whole remains

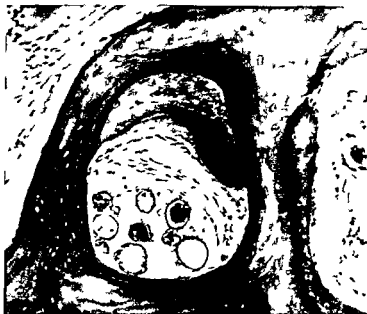


FIG 258—Revascularisation of dead bone. A cancellous space containing numerous newly formed thin walled vessels in its lower part. The upper part of the space has been filled in by a crescentic mass of new living bone showing well stained nuclei. The cell spaces in the surrounding dead bone trabeculae are devoid of nuclei. To the right of the figure is another deposit of new living bone forming a layer on the surface. The dead bone has in this case not suffered surface erosion prior to the deposition of the new bone.  $\times 100$

unchanged. A change has, however taken place in the revascularised focus, the centre of which shows a clear circle produced by osteoclastic absorption of the trabeculae. In the peripheral zone of this focus a high power view shows that there has been erosion of many of the dead bone trabeculae but that this has been made good and the structure further strengthened by newly formed trabeculae. This new bone can be clearly distinguished from the old because its nuclear staining shows it to be living

and because it is nearly all non lamellar (woven) bone (Fig 258)

The X ray aspects of the phenomenon of reorganisation of a focus of dead bone show (1) the development of clear areas or zones corresponding to the ingrowth of new vascularised tissue with resulting osteoclastic absorption of part or whole of the dead bone followed by (2) an increase in density due to new bone formation in these clear areas.

There is in some cases a further complication of the process which contributes to the X ray changes. In his studies of material from the above mentioned focal diseases of bone Axhausen has shown that a necrosed bone, although it may show no structural defect is less able to withstand pressure

than is living bone. On this account its trabeculae are apt to fracture and break up into small fragments that become compressed into a compact mass. In this way a portion of necrosed bone supporting part of a joint surface may suffer a depressed fracture with resulting deformity of the articular surface. In the case of necrosis of one of the small cancellous bones there may be a general collapse of the trabecular structure. Masses of compressed bone debris produced in this way are relatively resistant to the process of revascularisation and absorption and produce dense radiographic shadows (*vide* Fig. 403 (a) *et seq.*)

Aseptic necrosis of large masses of bone may occur in such conditions as tertiary syphilis and irradiation necrosis. Such masses may remain for a considerable time architecturally intact and therefore radiographically indistinguishable from the surrounding living bone. If secondary infection occurs e.g. with gummatous ulceration of the overlying tissues, a rapid osteoclastic erosion with separation of an unexpectedly large sequestrum takes place.

## XVI INFLAMMATORY CONDITIONS

Apart from the cellular changes in the soft tissues of the bone spaces and periosteum, which correspond with those seen in the various types of inflammation elsewhere, the bone itself may show (a) necrosis (b) osteoclastic erosion and (c) new formation.

(a) Necrosis.—Although bacterial or other toxins may produce bone necrosis there is little doubt that interference with the blood supply is the major factor in the production of the necroses in acute inflammatory conditions. In acute purulent inflammations in bone where pus is infiltrating the confined spaces in the bone or the deeper part of the periosteum increased pressure and damage to vessel walls is likely to produce thrombotic occlusion of all the small vessels in the affected area.

Caseous tuberculosis may lead to necrosis of bone in much the same way although in this case the toxins of the bacilli may also be an important factor.

(b) Osteoclasia.—Osteoclasia is a prominent feature of all inflammatory processes at the stage when granulation tissue is present. Numerous osteoclasts are formed at the surface of the granulation tissue in contact with the bone and a very active erosion occurs. Osteoclastic erosion being an active cellular process can take place only under conditions suitable for the activity of living cells, vascular granulation tissue provides such conditions.

FORMATION OF SEQUESTRA.—In the absence of an adequate blood supply, cellular activity ceases and bone erosion cannot take place. In areas of necrosed bone, the marrow spaces of which contain only dead tissue and leucocytes, osteoclasts cannot develop or function, and since leucocytes cannot erode it, the bone substance remains intact although the bone cells are dead. In the living tissue at the edge of the necrosed area a vascular granulation



tissue containing numerous osteoclasts develops and an active osteoclastic erosion of both living and dead bone occurs. This vascular granulation tends to extend into the spaces of the dead bone removing the necrotic tissues and dead leucocytes by means of macrophages and revascularising the area. Osteoclasts are developed in the advancing granulation tissue and in this way the dead bone may be completely removed. It not infrequently happens however particularly with large masses of dead bone that the granulation tissue fails to penetrate the whole mass. There is an active erosion on the surface and the necrotic area becomes separated from the living tissue as infection is still present leucocytes continue to emigrate from the capillaries and vessels of the surrounding granulation tissue. In this way there is formed an abscess cavity containing a portion of dead and partly eroded bone—a sequestrum. This sequestrum since it lies in a bath of pus separated from the living tissue undergoes no further erosion. Meanwhile the bone surrounded by granulation tissue in the wall of the abscess has undergone an extensive osteoclastic erosion. A radiogram taken at this stage shows therefore a sequestrum which is relatively dense in comparison with the surrounding zone of porosis.

The above account applies to pyogenic infections of bone. In tuberculosis the main feature is the development of granulomatous tissue. Osteoclasts are developed in relation to this tissue in the same way as with pyogenic granulation tissue but the process is less violent and advances more slowly. Necrosis (i.e. caseation) develops after the formation of granulomatous tissue which may have already resulted in considerable absorption of bone. Unabsorbed bone incorporated in a caseous mass does not undergo further absorption. A mass of caseous tuberculosis situated in a bone may of course undergo calcification as in other situations. Such calcification is in the form of an amorphous deposit.

(c) **New Bone Formation**—New bone formation is of course a prominent feature in inflammatory conditions and in most cases both woven and lamellar bone are formed at various stages of the process. Woven bone is produced during the more active stage and is gradually replaced by lamellar bone as the repair processes develop. The general histological features of this replacement have already been described. Woven bone as already noted is deposited in irregular trabeculae which can extend fairly rapidly throughout a mass of proliferating fibroblastic tissue derived from either periosteal or endosteal connective tissue. The bone formed in this way in a mass of soft tissue is not at first subjected to the normal strains and stresses of the part and the arrangement of the trabeculae appears to be determined by their relationship to the blood vessels passing into the tissue. Later when the mass of new bone begins to take the stresses and strains of the part its trabecular systems become orientated in relation to these forces. By this time much of the original woven bone has been removed and what

remains becomes enclosed and incorporated in trabeculae of lamellar bone. In this way a mass of bone of inflammatory origin formed outside the shaft of a long bone becomes converted into a new corticalis which is at first composed of longitudinally directed trabecular systems (in the form of sheets rather than rods of bone) separated by spaces containing vessels. The longitudinal spaces thus enclosed form new Haversian spaces with continued deposition of lamellar bone on the walls of these spaces Haversian systems form and a new compact corticalis is in time produced.

If we compare inflammatory reactions in bone with those in soft tissues it is clear that the stage of granulation tissue formation is characterised by marked osteoclastic erosion of the original lamellar bone with the formation of new woven bone while the stage of fibrosis is characterised by lamellar bone formation.

Conditions which would produce excessive fibrosis in soft tissues produce marked bony thickening with the deposition of considerable amounts of new lamellar bone. The marked fibrosis which takes place around a small enclosed abscess in the soft tissues has its counterpart in the continued deposition of lamellar bone which produces the marked bony thickening in relation to a similar abscess enclosed in bone.

## XVII EFFECTS OF TUMOURS ON BONE

The invasion of bone by either primary or secondary tumours commonly produces both bone absorption and new bone formation but in most cases bone absorption is the predominating result. It is relatively rare for bone formation to dominate the picture and produce an osteosclerotic reaction to tumour invasion. The striking absorption of bone which is commonly produced by an invading tumour has led to the belief that tumour cells are capable of eating away the bone matrix. There is however good evidence to show that the tumour cells themselves do not erode the bone but in some way stimulate an active erosion by the local osteoclasts. The mechanism of this stimulation remains uncertain. Two obvious possibilities are (a) the action of diffusible products from the tumour

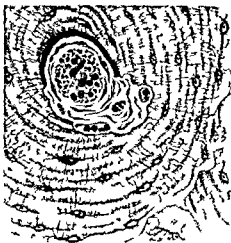


FIG. 209.—Part of a Haversian system with tumour cells (secondary carcinoma) in the Haversian vessel. The upper half of the circumference of the Haversian canal is intact and is bounded by a dark line of arrested growth; the lower half has been eroded by osteoclasts, two of which are seen in lacunae. Spindle cell endosteal connective tissue surrounds the vessel and fills the bay produced by osteoclastic action.  $\times 200$ .

cells or (b) alterations in such local conditions as blood supply or pressure within the bone spaces. Fig 259 illustrates erosion by osteoclasts in the neighbourhood of tumour cells. It is from a cross section of a femur showing extensive secondary carcinoma deposits in the form of emboli in the Haversian vessels, and also in places as masses of tumour cells in the medullary and Haversian spaces. A careful examination shows that active osteoclastic erosion takes place *before* the tumour cells come in contact with the bone. This is well seen in the figure, which shows inside the Haversian canal a small embolic mass of tumour cells surrounded by red blood corpuscles although there are no tumour cells in the perivascular space active osteoclastic erosion has already removed half the circumference of the Haversian canal and excavated an irregular cavity, this cavity is occupied by endosteal connective tissue. This appearance is seen in many places throughout this section and even when larger masses of tumour cells occupy eroded Haversian spaces a layer of spindle cells containing osteoclasts is found separating the tumour cells from the bone.

Such observations show that the invasion of the soft tissues of the bone spaces by tumour cells stimulates osteoclastic erosion which usually results in an extensive local porosis. In the later stages of this process the tumour masses may invade and occupy the eroded spaces in the bone and thus produce the impression that the tumour cells themselves have caused the erosion this, however is contradicted by studies of the earlier stages of the process. A benign tumour such as an enchondroma produces local absorption of bone in the same way by stimulating osteoclastic erosion.

In the case of primary bone tumours the mechanism of bone absorption is the same as that just described but in this case in addition to stimulation of the local osteoclasts there may be the possibility of the production of osteoclasts by the tumour cells. Such production is obvious in an osteoclastoma and in an osteosarcoma ( 'osteogenic sarcoma' ) the appearances often suggest that some of the tumour cells differentiate into multinucleated osteoclasts.

Osteosclerosis as a result of invasion of bone by tumour cells occurs only in a small proportion of cases, it is however, relatively common with metastases from certain types of growth, e.g. prostatic carcinoma. The rate of growth of the tumour cells is an important factor in determining this reaction, it occurs only with slow growing types of tumour such as produce a marked fibrotic reaction in the soft tissues. Metastases from slow growing scirrhous carcinomas of the breast or stomach may produce osteosclerosis.

The tumour deposits are visible to the naked eye as pale areas of dense bone replacing the cancellous structure. Involvement of the bone cortex with spread of the tumour cells to the periosteum results in the deposition of new bone on the surface of the original shaft. Microscopically the tumour deposits are in the form of small groups of cells lying in the Haversian canals or much restricted cancellous spaces within the bone. The tumour cells

produce at first some osteoclastic absorption of the bone, but this is followed by the formation of a considerable amount of new bone as the result of stimulation of the endosteal or periosteal connective-tissue cells. The spaces in the cancellous bone thus become filled in by deposits of new bone until only small central channels remain; these contain blood-vessels, endosteal connective tissue, and small groups of tumour cells. Much of the new bone produced is of the woven type, but lamellar bone may form and may cover woven-bone surfaces. This may occur in the new bone formed outside the shaft, so that irregular trabeculae of woven bone become converted into a new compact layer. In some very slow growing types of tumour, e.g. meningiomata invading the skull, a very dense thickening composed mainly of lamellar bone may be produced. A marked sclerosis of bone gives a dense, structureless radiographic shadow; moderate grades may produce more diffuse thickenings, which may closely resemble the appearances produced by Paget's disease.

In sclerosing types of osteogenic sarcoma much of the bone formation is the result of stimulation of the endosteal and periosteal tissues by the presence of the tumour, and is not the product of the tumour cells themselves. Secondary deposits of non-osseous origin involving the periosteum may result in irregular radiating outgrowths of woven bone similar to those seen in an osteogenic sarcoma. Deposits from adrenal neuroblastomas seem particularly apt to produce this effect.

### XVIII. EFFECT OF BLOOD-SUPPLY ON THE DEPOSITION AND ABSORPTION OF BONE

Much has been written on the effects of blood-supply on bone growth and on bone absorption and various "laws," e.g. "Hyperaemia causes bone absorption," have been stated. Unfortunately, the factors involved are complex and do not appear to operate according to such simple rules. There are, however, a few fundamental facts which can be stated at the outset.

(1) Since both bone deposition and bone absorption are active cellular processes requiring a supply of oxygen and nutritive fluids, neither can take place in the absence of a blood supply. On this account an infarcted area of bone (*q.v.*) remains structurally unchanged until revascularised.

(2) Where there is very active bone formation, e.g. in the metaphysis of a growing bone or in a healing fracture, an increased vascular supply is present.

(3) Active bone formation and active osteoclastic absorption are frequently found in progress together, e.g. in different parts of a single bone trabecula (Fig. 249); it is clear, therefore, that the same circulatory condition may favour both processes.

One of the fundamental difficulties in assessing the effects of an active hyperaemia arises from the fact that any form of increased cellular activity is associated with an increased blood-supply, and it is usually impossible to separate the two phenomena into cause and effect. It is clear, for instance,

in some cases that active cell growth determines the development of the increased blood supply—e.g. the extensively developed vascular supply of a tumour—at the same time growth could not continue without such increased vascular development. In spite of such difficulties evidence has been collected pointing to the fact that altered circulatory conditions may sometimes play a considerable part in determining the preponderance of either the deposition or the absorption of bone. An effect that appears to be a stimulation of bone growth secondary to hyperæmia is seen in some cases of increased growth in the length of a limb stimulated by traumatic or inflammation hyperæmia. Processes such as chronic osteomyelitis or joint tuberculosis in the region of the epiphysis may produce an increase in the rate of growth and ossification at the epiphyseal line. The focus of infection does not involve the epiphyseal line and appears to produce its effect by inducing a collateral hyperæmia (*Schubert* 1921). *Pommer* (1925) believed that osteoclastic activity is favoured by an increase in 'tissue pressure' in the medullary spaces of bones. Such increased pressure could be produced by vascular overfilling or by an increase in the cellular or fluid content of the soft tissues confined within the bone spaces. On this basis *Pommer* explained the active osteoclasia with resulting porosis found in neoplastic and inflammatory conditions in bone and also in some cases of local venous congestion. Acute post traumatic bone atrophy (*Sudeck's atrophy*) accompanied by local cyanosis and œdema is considered by *Pommer* (1925) and also by *Heydemann* (1933) to be due to an increased osteoclasia produced in this way. *Pommer* also considered that less acute types of atrophy following disuse are due not so much to the lack of stimulus to bone formation as to increased osteoclastic absorption, the result of venous stagnation. Since the venous return from the limbs is considerably facilitated by muscular movement inactivity tends to venous stagnation and hence increased venous pressure within the bone spaces. The beneficial action of massage in preventing inactivity effects could be explained on this basis. This 'tissue pressure' theory of *Pommer's* is probably the most comprehensive and elastic so far produced to explain various types of localised bone absorption. There are however many observations which are difficult to reconcile with it. For instance many cases have been recorded in which venous obstruction has resulted not in porosis but in a marked osteosclerosis. *Halshofer* (1937) records cases of diffuse sclerosis of the outer table of the skull associated with chronic obstruction of the superior vena cava resulting from pressure by an intrathoracic goitre in one case and by a large calcified lymph gland in another. *Pearse and Morton* (1930) produce what appears to be quite conclusive evidence that ligation of the deep veins from the part hastens the healing of a fractured fibula in the dog.

An earlier union with more solid callus was always found on the side with venous ligation. The clinical evidence of this effect of venous stasis brought forward by these workers is less convincing and raises the question whether

the changes in the circulation produced by various procedures are always in the direction intended. In the cases cited passive congestion was produced by compression of the limb from time to time by a sphygmomanometer bag. In the first of these cases which showed the clearest evidence of stimulation of callus formation this procedure resulted in an increased warmth in the affected foot. This seems to indicate that an increased *active* hyperæmia was the final result of the procedure which appeared to hasten callus formation.

It is certain that a marked diminution in arterial blood supply will retard bone formation. This is to be expected from what is known of the oxygen requirements of proliferating tissues and it has been shown experimentally in the case of fractures in a further study by the above workers. They also show that an experimental hyperæmia of one limb produced by a right lumbar sympathectomy resulted in only a slight increase in the rate of bone repair in three animals out of ten. In the remainder there was no effect at all (Pearse and Morton 1931).

It would not serve any useful purpose to discuss further the extensive literature bearing on the effects of circulatory changes in bone absorption and deposition. Sufficient instances have been given to show that the question remains unsettled and that much of the evidence produced is equivocal or contradictory. It is important to distinguish between active and passive hyperæmia but short of gross obstruction to the arterial supply it is not easy to be sure what circulatory changes are produced in the deeper tissues under various conditions or by a given procedure.

## XIX GENERAL FACTORS AFFECTING BONE ABSORPTION AND DEPOSITION THROUGHOUT THE SKELETON

So far we have considered some of the causes of local increase in absorption or deposition of bone. There are however several conditions in which derangements in these processes are generalised throughout the skeleton. The influence of the anterior lobe of the pituitary in increasing the size of the skeleton as a whole is shown in cases of gigantism and acromegaly.

*Paget's Osteitis Deformans*—In this condition there is a great alteration in the architecture of the bone which may affect practically the whole skeleton. Although the primary cause of this disease is unknown the cellular mechanism by which the bony changes are brought about is well understood. There is a continued excessive osteoclasia with an increased new bone formation that more than compensates in bulk for the bone lost. The process is slow and long continued and most of the new bone is lamellar in type. The excessive erosion disturbs the skeletal architecture however so that the compact bone becomes porous and replaced by irregular angular trabeculae which also form the cancellous bone. There is still an attempt at structural adaptation to stresses but this is very imperfectly achieved because the material is not used

(Bauer Albright, and Aub 1929 (b)) This continual loss of calcium must be derived from the bones and evidently corresponds to the normal osteoclastic erosion noted by Pommer as continuing throughout life. In estimating the calcium balance it is not practicable to starve the subject

but it is not difficult to construct a diet containing as little as 0.1 gm of calcium per diem while adequate in other respects. The usual procedure is to place the subject on such a low calcium diet and estimate the total calcium excreted in the urine and faeces over a series of 3 or 4-day periods. Under these conditions it is found that in normal adults the calcium loss exceeds the intake by about 0.15 gm daily. This gives a standard with which to compare the rate of loss of skeletal calcium under various abnormal conditions.

**Calcium Balance in Relation to Skeletal Changes**—It has been found that under the conditions of low calcium intake outlined above an output of calcium definitely in excess of the normal is always associated

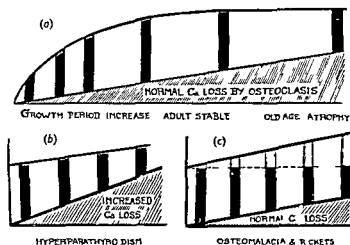


FIG. 210.—(a) Chart illustrating the balance between bone growth and bone absorption during life. The lower sloping straight line represents the steady calcium loss by osteoclasia which continues throughout life. The slope of the upper curved line represents the rate of growth during life. This is rapid in childhood and falls off during a full life while during old age it may cease altogether. The vertical bars represent the amount of skeletal tissue at various periods. During childhood growth greatly exceeds absorption and the amount of bone increases rapidly. In a full life these processes are balanced while in old age absorption is not made good by deposition of fresh bone and a senile bone atrophy results.

(b) Chart constructed in the same way as the last in a case of hyperparathyroidism. Here the greatly increased rate of calcium loss is shown by the steeper slope of the lower line. Bone deposition continues normally (upper line) but fails to keep pace with absorption so that the total amount of bone in the skeleton (vertical bars) diminishes.

(c) Similar chart in rickets or osteomalacia. Here the calcium loss (slope of lower line) is normal. The rate of bone formation (slope of upper line) is also normal but the bone formed during the disease fails to calcify and remains as osteoid tissue (shaded portion of bars). The result is a steady diminution in the amount of calcified tissue (lack of portion of bars) in the skeleton.

with some condition producing an increased osteoclastic activity throughout the skeleton. A purely local increase in osteoclasia does not appear to liberate enough calcium to produce a significant change in the calcium balance. This excessive output of calcium from the skeleton may or may not produce

a significant rise in the blood calcium. The excessive osteoclasia in hyperparathyroidism is associated with an increased calcium excretion, a raised blood calcium, and metastatic calcification in the soft tissues. A similar excessive osteoclasia occurs in some cases of hyperthyroidism; these show a marked increase in the calcium excretion rate but no changes in the blood calcium and no metastatic calcification (Hunter 1930). An excessive output of calcium from the skeleton associated with a marked general osteoclasia can be produced experimentally by the administration of parathormone or by very large doses of vitamin D. In both cases a raised blood calcium and metastatic calcification are produced. The mechanism by which the osteoclastic stimulation is produced is not understood. The results of parathormone excess and hypervitaminosis D are very similar and it has been suggested by some writers that vitamin D produces these effects by stimulating the parathyroids (Taylor, Weld, Brannon and Kay 1931). This has been disproved, however, by the discovery that the vitamin will produce the effects in parathyroidectomised animals (Shelling 1932, Dale, Marble and Marks 1932).

In rickets and osteomalacia there is no marked general increase in osteoclastic activity, and calcium balance experiments have shown that on a low calcium diet the rate of calcium excretion is within the normal range. It is clear, therefore, that the reduction in calcium content of the skeleton is not due to an abnormally rapid abstraction of calcium from the bone. This is further evidence against the existence of 'halisteresis' (Subsect XII). Studies on calcium metabolism are therefore in agreement with the conclusions based on histological findings and the factors involved in changes in skeletal calcium may be summarised as follows. A reduction in skeletal calcium may come about either by (1) an abnormally active osteoclastic absorption of bone, or (2) a failure to store calcium so that the calcium lost by the normal osteoclastic absorption is not made good. (2) Failure to store calcium may be due to either (a) failure to form bone, as in senile bone atrophy, or (b) failure in calcification of the bone matrix, as in rickets and osteomalacia. These points are illustrated diagrammatically in the charts (Fig. 260).

## REFERENCES

- VAHAUSEN, C. and BERGMAN, F. in: *Handbuch der speziellen pathologischen Anatomie und Histologie* (Ludw. Henke and Rössig) Berlin 1937 IX 3 118.  
 BAKER, S. L. and KAY, W. W. 1937 unpublished work.  
 BAUER, W., AICH, J. C. and ALBRICHT, F. (a) *J. exper. Med.* 1929 XLIX 1st (b) *Journ. Clin. Invest.* 1929 VII 70.  
 DALE, H., MARBLE, A. and MARKS, H. P. *Proc. Roy. Soc.* 1932 III 222.  
 VON JONSSER, V. *Sitzungsber. Akad. Wiss. Wein. Math.-naturwiss. Kl.* 1875 III 72 41.  
 EISENBACH, C. *Jena Z. Naturwiss.* 1864 I 347.  
 HALLSHOFER, I. in: *Handbuch der speziellen pathologischen Anatomie und Histologie* (Ludw. Henke and Rössig) Berlin 1937 IX 3 107.  
 HEIDEMANN, F. R. *Beitr. klin. Chir.* 1933 CLVII 561.  
 HOWSHIP, J. *Med. Chir. Trans.* 1819 X 176.  
 HUNTER, D. *Lancet* 1930 I 897, 947, 954.



- HUNTER, D, and TURNBULL, H M, *Brit J Surg*, 1931, XIX, 203-284
- KOELLIKER A, Die normale Resorption des Knochengewebes und ihr Bedeutung für die Entstehung der typischen Knochenformen,' Leipzig F C W Vogel, 1873
- KUMMER, R H, *Revue Med Suisse rom* 1917, XXXVII, 439
- LERICHE R and POLICARD A, 'Les Problemes de la Physiologie Normale et Pathologique de l'Os' Paris Masson et Cie edit, 1926, 'The Normal and Pathological Physiology of Bone,' London H Kimpton, 1928
- MAXWELL, J P, HU, C H, and TURNBULL H M, *J Path Bact*, 1932 XXXV 419
- PEARSE, H E, and MORTON H, *J Bone Jt Surg*, 1930, XII, 97, *ibid*, 1931, XIII, 68
- PETERSEN H in Handbuch der mik Anat d Mensch (W V MÖLLENDORFF) Berlin J Springer, 1930, II 2, 521-680
- POMMER G, Untersuchungen über Osteomalacie und Rachitis nebst Beiträgen zur Kenntnis der Knochenresorption und apposition in verschiedenen Altersperioden und der durchbohrnden Gefässe,' Leipzig F C W Vogel, 1885, *Deutsch Klin Chir* 1925 CXXXVI 1
- RETTERER, E *Journ l Anat et Physiol* 1906, XLII 193
- SCHLELLING, D H *J biol Chem*, 1932 XCVI, 215
- SCHNORL G, *Verh dtsh path Ges*, 1930 XXV, 205
- SCHUBERT A *Dtsch Z Chr*, 1921, CLXI, 80
- TAYLOR, N B, WELD C B, BRANION, H D, and KAY, H D, *Canad Med Ass J* 1931 XXIV 24 763 XXV, 20
- TOMES J and DE MORGAN, C, *Philos Trans*, 1853 CXLIII, 109
- WFIDENRICH, F in Handbuch der mik Anat d Mensch (MÖLLENDORFF), Berlin 1930 II 2

explanation of a sporadic as opposed to an hereditary defect would be forthcoming. This is however purely hypothetical and no actual explanation of the reason for a sporadic defect is at present available.

In those cases where the defects are hereditary in type many of them appear to follow the Mendelian law. In the main they appear as Mendelian dominants and in those families where the defects are present there appears to be little chance of their dying out in the course of a few generations. Definite information on this point is however difficult to obtain in most cases for whereas with Mendel's original experiments with peas and subsequent experiments on plants and animals it was possible to observe a large number of successive generations in the study of human inheritance no individual can possibly observe more than a few generations and in collecting family histories precise information is frequently very difficult to obtain for second hand information is extremely unreliable.

## CHAPTER XXI

### CONGENITAL MALFORMATIONS OF THE SKULL

#### CRANIOSTENOSIS

THE most important group of malformations of the skull is that which is due to a dystrophy of the membranous bones characterized by premature synostosis of one or more of the cranial sutures. The various skull deformities which arise from this cause are classed together under the general term of *craniostenosis* which includes such conditions as *oxycephaly*, *scaphocephaly*, *turmschadel*, hereditary *craniofacial dysostosis* of Crouzen and *plagiocephaly*. It seems probable that the condition described by *Engstler* as *Luckenschadel* is also most properly included under this heading.

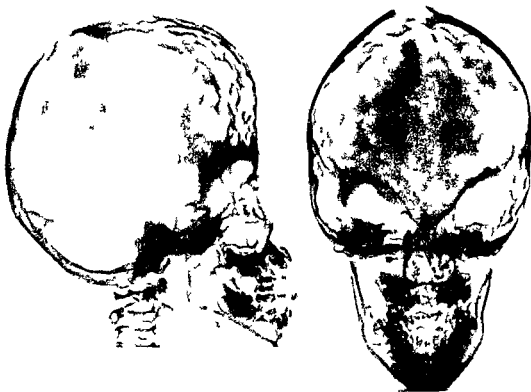
Various theories have been put forward to account for this premature closure of the cranial sutures. *Virchow* who first used the term *craniostenosis* regarded the condition as being inflammatory in origin. Since then syphilis and rickets have both been held responsible and a number of other different theories have been advanced to account for it. One of these was that the essential lesion in cases of *craniostenosis* is an abnormal development of the sphenoid bone but this is not the case. *Craniostenosis* is essentially a dystrophy of the membranous bones as opposed to a defective development of the cartilaginous base of the skull which produces the condition of *hypertelorism*.

The view put forward by *Reising* would appear to offer the most likely explanation of the cause of the condition. His view is that *craniostenosis* is a true congenital abnormality due to a variation in the germ plasma resulting in an unusual proximity of the ossific centres in adjacent bones and probably an abnormality also in the blastodermal matrix which normally separates the ossifying bones thus allowing this premature synostosis to occur. *Reising's* theory in addition to accounting for the deformities found in the skull would explain the abnormalities of development in other bones of the body which are sometimes found in association with the skull defects.

The general radiological features of the *craniostenosis* group of deformities are synostosis of some or all of the cranial sutures, deformity in the shape of the skull, well marked digital impressions in the much thinned cranial bones and changes in the base of the skull. Although the synostosis affects the cranial sutures in varying degree most authors agree that the sphenoccipital suture at the base remains open. In some cases there is internal hydrocephalus but this is not a constant feature and the radiographic appearances are not

those typically seen in hydrocephalus where the sutures are not closed and on the contrary may be widely open. Deformity of the skull which may not be obvious at birth develops as growth proceeds. It must be realised however that definite craniostenosis may be present in the absence of any gross change in the shape of the skull if all the sutures are affected and all close together.

The clinical manifestations of the condition are deformity of the skull



FIGS. 61 and 62. Craniostenosis. Oxycephalic type. Note the marked cranial moulding, the acute basal angle and the increased vertical diameter of the skull.

which varies very much in extent, exophthalmos and sometimes divergent strabismus. Headache usually occurs and loss of vision may result from optic atrophy. It is said that loss of smell and hearing may also occur, but there is some doubt if craniostenosis produces these effects.

The type of skull deformity produced by craniostenosis varies according to which of the sutures are affected.

Oxycephaly is the type of deformity most commonly seen and produces a very obvious change in the patient's appearance (Figs. 261 and 262). The skull is short from front to back and its vertical diameter is increased, producing a

very characteristic appearance to which the name of tower skull has been applied. Grieg says that in its slightest form it attracts attention while in its grosser forms there is no passer by but is shocked by the disfigurement and repelled by its hideousness. As with all types of craniostenosis there is a marked hereditary tendency. This type of deformity has been classified as (1) *true oxycephaly* (2) *delayed oxycephaly* and (3) *pseudo oxycephaly* but it seems probable that true and delayed oxycephaly only differ in degree and in the rate at which the sutures close. Pseudo oxycephaly is not a craniostenosis but refers to a skull of ab normal shape which approximates to that seen in oxycephaly. Grieg says that in true oxycephaly the skull is sutureless but this is not borne out by other observers. Sear is very emphatic that in children with definite craniostenosis of the oxycephalic type the basal suture between the sphenoid and the occiput may be widely open and the writer's experience endorses this view (Fig 263).



FIG 263.—Craniostenosis, showing the entire absence of cranial sutures but the open sphenoid-occipital suture at the base.

The deformity is caused by the rapid growth of the brain in childhood. The premature closure of the sutures prevents the normal symmetrical expansion of the cranial cavity and those parts which can yield do so. The cranium bulges upwards usually towards the anterior fontanelle and as a rule its highest point is at the bregma but it may vary in position anywhere from the anterior to the posterior fontanelle. Lateral bulges may also occur in the region of the temporal and sphenoidal fontanelles. The usual type of oxycephalic skull shows an almost vertical extension of the cranial cavity rounded in its upper part but the shape varies in different cases. Sometimes the upper part of the skull becomes almost pointed—the *acrocephalic* skull. The soft base of the skull also yields under the increased pressure and the bulging forwards of the middle fossa produces a decreased

depth of the orbital cavities which become so shallow that the eye are pushed forward producing the hideous exophthalmos which is typical of a marked degree of oxycephaly. As a rule there is no enlargement of the sella turcica although this has been described in some cases. The cranial bones are thinned—usually markedly so—and moulding is pronounced so much so that in some cases the appearance approximates to that seen in Luckenschadel.

**Scaphocephaly** is the type of deformity which results from premature closure of the sagittal suture while the other sutures remain open. Instead of increased height seen in oxycephaly the skull is increased in length from front to back and in place of the temporal bulging the skull is narrowed. While in oxycephaly the basal angle is more acute than normal in scaphocephaly it is flattened. Exophthalmos does not occur and as a general rule there is little cranial moulding. Most cases of scaphocephaly are of little importance and cause no disability apart from the unusual shape of the skull.

**Plagiocephaly** is the name given to asymmetrical development of the skull produced by unilateral craniostenosis—usually at the temporo parietal suture. It is obvious that different types of asymmetrical development of the skull may occur in craniostenosis which vary according to which of the sutures are involved and that combinations of the more usual types of deformity may occur.

### HEREDITARY CRANIOFACIAL DYSOSTOSIS OF CROUZEN

In 1912 Crouzen described a craniofacial deformity due to synostosis of the skull bones. The appearance of the skull is similar to that seen in oxycephaly but the facial changes are more marked. The superior maxillæ are atrophied the nose has a wide bulge and is curved like a parrot's beak. The lower jaw shows a condition of prognathos. There seems to be no adequate reason for separating this condition from oxycephaly of which it would appear to be a type. As in the case of oxycephaly it may or may not be hereditary.

### LUCKENSCHADEL

This is a rare congenital skull deformity first described by *Engstler*. The radiogram shows an extraordinary appearance of the cranium which appears to consist of a network of bony ridges surrounding bony defects (Fig. 264). The normal cranial sutures are absent. Actually in the areas from which the bone appears to be missing there may be a thin layer of bone but in other cases there is no bone present and in these areas the cranial wall consists only of a thick membrane. Opinions are divided as to the true nature of this condition but it would appear to be due to an extreme degree of craniostenosis.

## ACROCEPHALY-SYNDACTYLY

The association of skull deformities with certain cases of syndactyly has long been recognised and in 1906 *Apert* introduced the term *acrocephaly syndactyly* to describe these cases but here again there appears to be no reason to separate this group from the general group of *craniostenoses*. As previously mentioned *Reaping's* theory as to the causation of *craniostenosis* is interesting inasmuch as it would account for the group of cases described by *Apert*.



FIG. 264.—Lückenschädel

## HYPERTELORISM

This is the name applied to a skull deformity which was previously included as a type of *oxycephaly* until it was separated from that condition by *Grieg*. It is characterised by a wide separation of the eyes caused by an unusually wide nasal bridge. Whereas *craniostenosis* is essentially a dystrophy of the membranous bones of the cranium *hypertelorism* is due to an abnormal growth of the cartilaginous base of the skull. The sphenoid shows an abnormal development of that portion of it which is developed in cartilage. The greater wings are abnormally small and the lesser wings abnormally large with the consequence that the frontal bones are pushed upwards, outwards and forwards producing an interfrontal groove. The nasal bones are abnormally broad and an internasal bone is present between them in the internasal suture. The facial appearance which results from these deformities is characteristic and shows a



I ro 263



I ro 67



I ro 008

I ro 005 000 an l 07—llo o l t a y e l o c a a l l y s c o s t o s s s h o w n g c o m p l e t e l e n c o f l o t l l a c l e s t l a r g e  
 u n o u s f l e l n c a s i n t l o e l l a l l o n o e f e c t a a l s o t l o r e g o o f t l o s y m p l y s s p l b s



low forehead with pronounced frontal eminences a broad depressed nasal bridge, widely separated eyes, and an external squint

### HEREDITARY CLEIDO-CRANIAL DYSOSTOSIS

This is a rare congenital deformity which has been regarded as a defect in the ossification of membranous bone, and changes are certainly seen in the clavicles and the skull in many cases, but *Fitchett*, who has made an investigation of this condition, regards it as being primarily a congenital defect of the development of the clavicles analogous to similar defects in other bones, such as the radius and the fibula. He points out that not only that portion of the bone which develops in membrane but the whole of the bone may be absent, and that while in certain cases there are skull changes, in others there are none. He considers that the condition should be described as congenital cleido dysostosis with or without other developmental anomalies (Figs 265 266 and 267)

The defect in the clavicles varies in extent in different cases, the condition most frequently seen being an absence of the central portion of the bone. In some cases the sternal end only of the bone is present and very occasionally the acromial end only. Both clavicles may be completely absent.

Skull changes are usually present also and large unossified areas may be seen. The fontanelles and sutures are widely open and the anterior and posterior fontanelles may be united in one large bone defect extending from the front to the back of the skull. The frontal and parietal eminences are unduly prominent, and the bones show much bossing.

The bones of the face are small, as are also the nasal sinuses, and the base of the skull is short. Dentition is delayed.

As in the case of all congenital defects, associated deformities may be found in other parts of the skeleton.

Clinically, the patients show a large head and a small face, but the most striking feature is the patient's ability to bring his shoulders together to the middle line. The condition shows a marked hereditary tendency, but isolated cases are recorded.

## CHAPTER XXII

### CONGENITAL MALFORMATIONS OF THE SPINE AND PELVIS

THE CONGENITAL abnormalities of the spine vary from such gross deformity as is seen in a case of myelocoele which is incompatible with life to the minor variations in the lumbo sacral region classified as *spina bifida occulta* which is disclosed only as a result of a radiographic examination and occurs so commonly as to make it difficult to decide whether it would not more properly be described as a normal variation.

The normal arrangement of the vertebrae is seven cervical twelve dorsal five lumbar five sacral and five coccygeal. The total number is fairly constant and the simplest variation is what is known as pre or post fixation of the thorax. In pre fixation the rib bearing vertebrae move up one and ribs are found associated with the seventh to the eighteenth vertebrae while in post fixation the rib bearing vertebrae move down one and there are consequently eight cervical and four lumbar vertebrae. There may however be an extra rib bearing vertebra in either the cervical or the lumbar region or the last rib may be absent on both sides. In addition to these symmetrical variations there may be an extra rib or ribs on one side or a unilateral deficiency in the number of ribs. These variations are associated either with supernumerary or deficient hemivertebrae.

The most common sites for variations in the development of the spine are in the atlanto occipital region and in the regions of transition from cervical to dorsal dorsal to lumbar and lumbar to sacral vertebrae. The vertebrae are normally ossified from three primary centres—one for the body and one for each side of the neural arch. The centres for the neural arch unite posteriorly in the first year of life and the neural arch unites with the body in the third year. Variations in these ossific centres and failures of the component parts of the vertebrae to unite normally occur in certain cases with resulting deformities which vary greatly in degree. The body instead of ossifying from one centre may ossify from two separate ossific centres and if these fail to unite there is a resulting anterior *spina bifida* or a centre may appear for one half of the vertebral body without a corresponding one for the other half in which case we have a hemivertebra. If this occurs in the dorsal region the rib which would normally correspond with the missing half is also missing. There may also be supernumerary hemivertebrae and if these occur in the dorsal region there will be corresponding supernumerary ribs. In the same way the centres for the neural arch may fail to unite and produce a *spina bifida*.

but in addition to these simple variations we may find gross congenital abnormalities in which an extremely irregular bone formation occurs in the affected area with the result that the normal structure of the spine is entirely lost

### CERVICAL REGION

It has already been mentioned that the congenital abnormalities tend to occur most frequently in the transitional regions of the spine and as we consider the spine from above downwards the first type of variation occurs in the upper cervical region. Here various types of synostosis occur. The atlas



FIGS. 288 and 289—Klippel-Feil Syndrome. The radiograms show a short neck with the combination of fused vertebrae and spina bifida.

may be fused with the occiput—occipitalisation of the atlas—the atlas and the axis may be fused together or the odontoid may be fused to the atlas.

**Klippel-Feil Syndrome**—*Klippel* and *Feil* first described an abnormality of the cervical spine which is associated with their names. Patients with this condition show a shortening of the neck with marked limitation of movement in this region (Figs. 288 and 289). They also have hair growing low down into the neck. When examined radiographically—a procedure which is not at all easy to carry out satisfactorily owing to the fixation of the head and the frequently associated torticollis—it is found that several vertebrae in the upper cervical region are fused together. Sometimes the fusion is complete but in some cases it may only affect the vertebral bodies and in others only the neural arches. Below the fused vertebrae there is usually a spina bifida.

affecting the lower cervical vertebræ which show a failure of closure in the arches posteriorly

It may be mentioned here that although for convenience the various types of congenital abnormality are considered as they occur in the different regions of the spine they do not necessarily occur singly and a variation in one region may be and frequently is associated with one or more abnormalities in other parts of the spine—or in fact in other parts of the body—and the Klippel Feil syndrome is no exception to this

**Cervical Rib**—In the lower cervical region the most important variation is the presence of a rib bearing seventh cervical vertebra. The cervical rib may be unilateral or bilateral. If unilateral it may be associated with a supernumerary hemivertebra but as a general rule a unilateral cervical rib occurs in association with an otherwise normal seventh cervical vertebra. A cervical rib may be very rudimentary or it may be a well formed rib which articulates at its anterior end with some point on the first dorsal rib. The presence of a cervical rib is important because it may cause pressure on the nerves of the brachial plexus. In this connection it must be remembered that it is not necessarily the longest or the best formed rib which is most likely to give trouble. While it is true that a well formed cervical rib may cause pressure on the nerves one often finds ribs of this type which are easily palpable in the neck causing no untoward symptoms whatever. On the other hand a rudimentary rib which on the radiogram appears to be insignificant may be the cause of a severe nerve lesion for rudimentary ribs of this type are frequently connected to the first dorsal rib by a dense band of fibrous tissue which although invisible on a radiogram may be and frequently is a definite source of pressure on the nerves.

Although accessory ribs in the cervical region are almost always found in connection with the seventh cervical vertebra cases have been described showing cervical ribs associated with the fifth and sixth cervical vertebræ—such cases are however rare. Without the actual development of a cervical rib the costal element of the seventh cervical vertebra may show considerable enlargement from which as from a cervical rib a dense fibrous band may extend to the first dorsal rib and cause similar symptoms of nerve pressure.

## DORSAL REGION

Congenital abnormalities of the dorsal region of the spine and of the ribs must be considered together since a vertebra and its corresponding ribs constitute a spinal segment. As mentioned above the whole thoracic cage may be moved upwards or downwards the first rib bearing vertebra may be the seventh or the ninth and the last the eighteenth or the twentieth. There may be only eleven rib bearing vertebræ or there may be thirteen. Deficiency of the ribs may be bilateral or unilateral and the same applies to extra ribs. Cases of gross abnormality of structure of the dorsal spine will also show gro

rib deformities (Fig 270) Several ribs on one or both sides may be absent, or may show irregular formation with extensive fusion. A case of simple hemivertebra, whether superior, numerary or not, will show a rib only on the side of the half vertebra. Apart from numerical variations and gross deformity, one often finds a simple rib variation in which the anterior end of a rib is bifid or splaved out. While many of the spinal deformities are only of academic interest, the bearing of spinal deformities on scoliosis is of considerable importance. Congenital scoliosis is except in rare cases due to congenital deformity of the spine—in the dorsal region usually due to a hemivertebra. It is important to realise that although the scoliosis is due to a congenital defect, it will in all probability not be discovered until the child sits up or walks, and in some cases it may not be discovered until adolescence. For although it used to be thought that all cases of scoliosis developing in adolescence were of the static type, radiographic examination shows that some of these cases are due to a previously unrecognised spinal deformity.



FIG. 0.—Gross congenital abnormality of the dorsal spine with corresponding abnormalities in the ribs.

## LUMBAR AND SACRAL REGIONS

In the lumbar region there may be an extra vertebra, or one may be deficient. Lumbar ribs may be present. The most important variations, however, occur at the lumbosacral junction. The fifth lumbar vertebra may be joined to the sacrum, a condition known as *sacralisation of the fifth lumbar vertebra*. This sacralisation may be unilateral or bilateral, and in the unilateral cases we have one of the causes of congenital scoliosis. Union of two of the lumbar vertebrae may occur, and it must be remembered when considering a radiogram of an adult patient which shows fusion of two of the

lumbar vertebræ that old-standing disease of the vertebral bodies is not the only cause of the condition

**Spina Bifida**—Spina bifida may occur in any part of the spinal column but the majority of cases occur in the lumbo sacral region. It is due to a defective development of the spinal canal and according as the normal development is arrested at various stages so the different types of spina bifida are produced varying from myelocele or rachischisis to minor cases of spina bifida occulta. Of the five varieties described *myelocele* is incompatible with life. Its interest for the radiologist however lies in the fact that it may be recognised *in utero* at a pregnancy examination by a localised kyphotic deformity in the lower part of the foetal spine which extends over several of the vertebral bodies. The appearance seen is quite characteristic and its recognition is of course valuable information for the obstetrician (*vide* Volume II Fig 294). *Syringomyelocele* and *meningomyelocele* are due to gross changes in the spinal cord and the cauda equina and are associated with paralysis for which treatment is of no avail. The only types which are of importance inasmuch as that treatment is possible are *meningocele* and *spina bifida occulta*. In *meningocele* no true nerve tissue is involved the cord and the nerves arising from it are normal and the tumour is due to the distended meninges bulging through a defect in the neural arch due to arrested development of the laminae and their failure to unite posteriorly. Such a sac may in suitable cases be removed.

In spina bifida occulta there may or may not be any symptoms. Paralytic symptoms when present are usually due to adhesions and surgical treatment may in such cases effect a cure. Radiographic examination in these cases is of the greatest importance for it may be the only method of establishing the diagnosis with certainty. The fifth lumbar vertebra and more frequently the first sacral vertebra are the ones which most commonly show a condition of spina bifida occulta and anomalies of the development of the arch of each of these vertebræ are extremely common. Failure of the laminae to unite posteriorly may be seen as a simple fissure or the ununited portions of the arch may run obliquely at different levels and the spinous process may also be obliquely placed. Again the spinous process of the first sacral segment may be absent leaving an open space approximately triangular in shape—the so-called open sacrum. It is extremely difficult to decide how far these variations are to be regarded as abnormalities or at least since they are all abnormal how far they are to be regarded as being of any significance. The more severe types when the lesion affects several vertebræ are obviously definitely abnormal but simple failure of fusion and fissure formation and the various degrees of obliquity seen in the arrangement of the ununited portions of the arch of the first sacral segment are seen so frequently as accidental findings in the course of an examination for some other purpose that it is difficult to assess their clinical importance. A very large number of apparently normal people when examined radiographically show a spina bifida occulta on the other

hand spina bifida occulta may occur in others in association with signs and symptoms which indicate its presence. In some cases paralysis of the lower limbs, neurotrophic disturbances, sphincter troubles, nocturnal enuresis, and gradually developing deformities of the feet may occur, and these may be accompanied by such local signs as depression of the overlying skin, the presence of a tuft of hair, or an area of pigmentation or telangiectasis. There fore, while a simple fissure or minor bone defect may be of no clinical significance, it is important to examine radiographically all patients, particularly children, who show such troubles as those mentioned.

## THE PELVIS

The pelvis is rarely the seat of marked degrees of congenital abnormality. The deformities of the sacrum already mentioned constitute the commonest type of deformity seen. Two rare types of deformity call for mention, however, on account of their importance in obstetrics. The Negele and the Robert type of pelvis are respectively the unilateral and the bilateral type of the same congenital defect, and are due to defective development of the ala sacri on one side in the case of the Negele pelvis, and on both sides in the Robert pelvis.

In the Negele pelvis one wing of the sacrum is either absent or rudimentary, the os innominatum on the affected side is smaller than normal, and the sacrum and the ilium are fused together at the sacro iliac joint. The result of this deformity is to produce a marked obliquity of the pelvis with the symphysis pubis over to the sound side.

In the Robert pelvis, the rarest type of deformed pelvis from the obstetrical point of view, the defect is symmetrical. Both alae sacri are affected, and instead of an oblique pelvis a symmetrical narrow pelvis is produced.

There has been a considerable amount of dispute as to whether these pelvic deformities are due to a congenital defect or to old standing disease at the sacro iliac joint, but the evidence points conclusively to the fact that in the true Negele or Robert pelvis there is no sign of a previous inflammatory lesion. The confusion appears to have arisen because in either type of defect the sacro iliac joint is obliterated, but such fusion of bones in the case of congenital abnormalities is, of course, a common experience in other parts of the skeleton, and is seen especially frequently in spinal defects. A pseudo Negele or pseudo Robert pelvis may, however, be produced in cases of old osteitis of the sacro iliac joint on one or both sides.

## CHAPTER XXIII

### CONGENITAL MALFORMATIONS OF THE UPPER AND LOWER LIMBS

THE EXTREMITIES may show many congenital deformities varying widely in extent. They may fail to develop entirely or may become mere stubs of limbs. Sometimes the proximal segment of the limb is normal and the distal portion is deficient—*hemimelus*—or conversely the proximal segment may be missing and the hand or foot may spring directly from the trunk like a seal's flapper—*phocomelus*. The lower limbs may be fused together throughout their entire length with varying bony defects and synostosis producing the mermaid types of deformity. In such cases of gross deformity a radiogram will demonstrate the nature of the bony abnormality.

### THE UPPER EXTREMITY

#### The Shoulder Girdle

**Sprengel's Deformity**—This is a congenital deformity due to a failure of the scapula to descend from its embryonic position in the neck to the upper part of the thorax. It is therefore abnormally high in position. As a rule it is



FIG. 71.—*Sprengel's Deformity*. The scapula on the affected side is high in position and is associated with abnormal development of the cervical vertebrae; and the presence of a plate of bone extending outwards from the affected side to the scapula.



rotated so that the inferior angle is nearer to the spine than the superior one but the rotation may occur in the opposite direction. The bone is abnormal in shape and tends to approximate more nearly to that of an equilateral triangle instead of having the vertebral border longer than the axillary one. It is concave with its concavity forwards owing to the bending forward of the suprascapular portion of the bone. The superior angle is frequently opened out producing an angulation in the vertebral border. From the upper part of this border or from the superior angle there may be a fibrous band uniting the scapula to the spine or there may be an extra bone or cartilaginous plate in this position which corresponds with the suprascapula bone of the lower animals (Fig. 271). If such a supernumerary bone is present it is usually triangular in shape with its base adjacent to the upper part of the vertebral border of the scapula and its apex against one of the lower four cervical vertebrae. It may be united to the spine or the scapula by cartilage or there may be bony union at either end thus firmly uniting the scapula to the vertebral column by a continuous bridge of bone.

In some cases there is also an associated cervical spur bifida or a hemi vertebra. In its simplest form Sprengel's deformity consists of nothing more than a scapula which is placed unusually high in position and shows little variation in shape from the normal while in its extreme form there is in addition to the elevation of the scapula deformity of the bone and gross congenital defect in the cervical vertebrae. Between these two extremes varying degrees of deformity are found showing one or more of the abnormalities described above. The condition may be bilateral or unilateral but the unilateral form is the one more commonly seen.

Hereditary Cleido-cranial Dysostosis has already been described in connection with skull deformities.

### The Humerus

The bones of the arm are of course completely missing in cases where the child is born without one or other of the arms and as already mentioned cases are recorded in which the humerus and radius and ulna are missing and the hand articulates directly with the scapula. Apart from such cases however it is extremely rare to find a congenital absence of the humerus. In fact the humerus shows little tendency to congenital variation. Cases have been described where the shoulder joint shows a congenital abnormality in which the glenoid is convex and the head of the humerus concave but they are extremely rare. The congenital deformities in both upper and lower limbs increase in number as one passes towards their distal ends.

### The Radius and Ulna

Of the long bones of the arm the radius is the one which is most commonly absent or partially so for in cases in which clinically the radius appears to be absent a radiogram frequently shows that the proximal end of the bone is

present. Other cases are described in which both ends of the bone are present and the middle portion is missing. Associated with congenital absence of the radius there may be congenital absence of the thumb and sometimes also of the fingers on the radial side of the hand (Fig. 272).



FIG. 272. Congenital absence of the radius with associated absence of one metacarpal bone and the bones of the corresponding finger.

Congenital absence of the ulna may occur but such cases are rare as an isolated defect. Here again the defect may be complete or partial. Congenital absence of either the radius or the ulna or of either of their distal extremities produces what is known as *congenital club hand* in which condition the hand is deviated as would be expected towards the side from which the bone is missing and either a radial or an ulnar club hand may be produced accordingly.

Synostosis of the bones of the forearm may occur most commonly at the proximal end of these bones and there may be an associated dislocation of the head of the radius (Fig. 273). Such cases of synostosis show a marked hereditary tendency. In addition to the synostosis of the radius and ulna cases have been described in which the humerus, radius and ulna have all been fused at the elbow.

A rare congenital deformity which occurs at the elbow joint is what has been described as *patella cubiti* in which the upper extremity of the ulna is deformed and a large sesamoid bone is found on the dorsal aspect of the elbow joint analogous to the patella at the knee joint.

### The Hand

The congenital deformities of the hand are very numerous and show a large number of variations. There may be supernumerary digits or one or more digits may be absent. Phalanges may be missing or deformed. One metacarpal bone may be absent; its neighbour may show a forked distal extremity supporting two fingers; or the first metacarpal bone may support two thumbs. There is an almost endless number of variations from minor defects to complete disorganisation of the normal architecture of the hand (Fig. 274).



FIG. 273.—Synostosis at the upper end of the radius and ulna.

Certain well recognised types of deformity may, however, be mentioned. *Brachyphalangia* is a condition in which there is an abnormal shortness of one or more of the phalanges, usually the middle one. In *brachydactylia* the metacarpals are abnormally short, or this shortness may affect only one of the metacarpals in which case it is usually the fourth which is affected. A gross deformity in which the hand is more or less split into two parts is known as the *lobster-claw deformity* (Fig. 275). In this the fingers are fused into a radial and an ulnar group and one or more of the metacarpals is missing or misplaced. *Syndactyly* is a condition in which the fingers are webbed. It may affect two or more fingers and the union may be by skin only, or by skin and connective

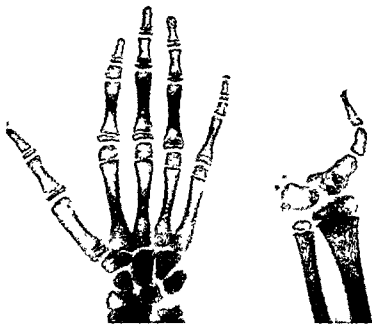


FIG. 274.—Gross congenital abnormality of one hand in which the bones show a 'foot type' of development. Notice also the brachyphalangia in the index and little fingers of the other hand.

tissue, or the bones may actually be fused. *Polydactyly* or the presence of extra digits or parts of digits, is a fairly frequent abnormality, and as many as eight digits may be found on one hand. The extra digits usually occur at one or other end of the row, although centrally placed extra digits may be found. There is usually little difficulty in deciding radiographically which are the extra digits for they are as a rule, abnormally developed. All these congenital deformities of the hand may be unilateral or bilateral and they show a very marked hereditary tendency. Brachydactyly, syndactyly, and polydactyly especially are very frequently inherited and may be traced through many generations.

*Arachnodactyly* (spider fingers) is the name given to a condition in which the fingers and toes are unduly long and slender. It is a rare condition which was first described by *Marfan* in 1896 since when only a few cases have been recorded. The case illustrated is the only one the writer has seen (Fig 276). A number of causes have been suggested to account for the condition but it is



FIG 275 Lobster claw deformity of the hand



FIG 276 Arachnodactyly

now regarded as a congenital abnormality a view which is supported by the fact that in the recorded cases the abnormality in the hands and feet is associated with other congenital defects. The long bones generally are increased in length and children with this condition are tall for their age. Skull defects may be present and ocular symptoms due to dislocation of the lens and trembling irides have been described. The muscles are weak and atonic and it has been suggested that the condition is allied to amyotonia congenita.

### THE LOWER EXTREMITY

The congenital deformities of the lower extremity vary from complete absence of both limbs to minor variations in the development of the phalanges. The presence of three lower limbs has been described. As in the case of the arms the variations occur most commonly in the distal portion of the lower limbs.

#### The Femur

The femur may be absent in one or both legs, or more commonly it may be partially absent in which case either the upper, middle, or lower portion of the bone may be missing. When the upper and middle portions are missing the lower end may be fused to the tibia.

#### The Tibia and Fibula

The commonest gross congenital deformity of the leg bones is absence of the fibula. More rarely there may be congenital absence of the tibia. In either case the defect may only be partial. When the tibia is absent, the patella may also be missing. Congenital absence of either the tibia or the fibula is one of the causes of congenital club foot.

#### The Foot

As in the case of the upper limb the bones of the hand are the commonest site of congenital abnormalities so in the lower limb the bones of the foot are most commonly affected. One or more bones may be missing and *polydactyly*, *syndactyly*, *arachnodactyly*, and *brachydactyly* occur as they do in the hand. In the tarsus two or more of the bones may be fused. Deformities of this type are not so important in the foot as they are in the hand but of course, gross deformities in the lower limb may constitute a serious handicap.

### CONGENITAL AMPUTATIONS

These may occur in the limbs at any level. In the past they have been ascribed to a variety of causes but in general the cause has been assumed to be some intrauterine constricting band which may produce a congenital furrow round the affected limb or even produce a complete amputation. Amniotic bands and constriction by the umbilical cord have both been regarded as possible causes but more recent work casts very considerable doubt on the theory that either of these factors is ever the real cause, which appears to be an intrinsic one.

### CONGENITAL DISLOCATIONS

Congenital dislocations may occur at several joints, but the only one which is at all commonly seen is that which occurs at the hip joint. Congenital

dislocation at the knee joint occurs next in order of frequency, but it is a rare condition, and while congenital dislocations have also been described at the shoulder, elbow, wrist, and ankle, and also in the small joints of the fingers, they are so rare as to be curiosities of the literature on this subject

### **Congenital Dislocation at the Shoulder Joint**

A congenital dislocation at this joint is very rarely seen. It must be distinguished from a dislocation due to paralysis or to trauma at birth. It is usually associated with congenital bone defects at the joint.

### **Congenital Dislocation at the Elbow Joint**

This again is a very rare condition, and generally consists of a subluxation of the head of the radius. The more severe forms are associated with bone defects such as synostosis of the radius and ulna.

### **Congenital Dislocation at the Hip Joint**

This is an extremely important congenital deformity from the radiologist's point of view, inasmuch as success in treatment depends in a great measure on



FIG. 977.—Congenital dislocation of the left hip joint, with poor development of the ossific centre of the femoral head.

its early recognition. It may be either bilateral or unilateral, the unilateral cases occurring about twice as commonly as the bilateral ones. It exists at birth, but as a general rule is not recognised until the child begins to walk. The dislocation may be complete, or there may be only a subluxation at the hip.

joint. The displacement of the femur, if only slight, may easily be overlooked in an infant, since there is little of the bone at the upper end of the femur ossified. It is therefore essential to obtain a radiogram with the pelvis and limbs correctly placed in the straight position. Before the femoral epiphysis is ossified, the abnormal shape of the acetabulum is the most important point in the recognition of the condition, but after the epiphysis has appeared, the dislocation is more easily recognised. The epiphysis is smaller than normal, and ossification is delayed. In unilateral cases the delay in the appearance of



FIG. 278.—Congenital dislocation at the hip joint. Note the lack of development of the acetabulum and the small epiphysis of the head of the femur on the affected side.

the epiphysis can easily be recognised, as can also its smaller size (Figs. 277 and 278).

Opinions differ as to the cause of the condition. Some writers maintain that it is due to a defect in the development of the acetabulum, where the normal bony outgrowth which produces its outer margin or rim is deficient, and that it is therefore primarily a bone defect. On the other hand, there is the view that the primary defect is an abnormal slackness of the ligaments and tendons, and that the bony changes are a secondary development—and certainly in support of this view we have the fact that if the condition is recognised and treated at an early stage, the acetabulum appears to develop normally after the dislocation has been reduced.

At birth the whole of the upper end of the femur, including the neck and the trochanters, is cartilaginous, as is also a good deal of the acetabulum. At this stage there is no evidence of any defective development of the acetabulum, but as growth proceeds, the acetabulum is seen to be abnormal, for whereas normally

the upper part of the acetabulum is placed horizontally and makes a buttress which prevents upward movement of the femoral head in a case of congenital dislocation this upper portion is much reduced in size or absent. The acetabular cavity is shallow and tends to come almost into line with the outer surface of the ilium and in fact does so except for a small projection which is all that is developed of the normal projecting upper margin. The head of the femur is normal at birth but secondary changes occur later. As it does not have to carry the body weight or withstand the normal stresses which are the result of its position in close apposition to the acetabulum ossification is delayed and in unilateral cases a marked degree of difference may be seen in the size of the ossified femoral epiphysis on each side. In the absence of the femoral head the already shallow acetabulum becomes more markedly so by the overgrowth of cartilage and fibrous tissue a point which may be recognised radiographically by the wide joint space seen after reduction has been effected. In old standing cases as the femoral head becomes fully developed it becomes conical in shape and the neck of the femur which is short is placed horizontally and twisted forwards it is therefore seen foreshortened in a radiogram. The pressure of the misplaced head of the femur against the side of the ilium may produce a depression in the bone which constitutes a false acetabulum. In most cases this depression is quite shallow but in some cases it may become quite a well formed socket in which the femoral head is held securely (Fig 279).

The diagnosis of the condition is perfectly obvious radiographically when the child is not seen until after the epiphysis at the head of the femur has become ossified but when seen at an earlier stage its recognition is difficult. There is no abnormality to be seen in that portion of the acetabulum which is ossified and the only guide is the displacement of the femur. This varies greatly in extent. At first there may be no recognisable displacement in fact displacement may be negligible until weight bearing begins. If there is any displacement it may be recognised by the break in the normal continuous curve from the neck of the femur to the pubic ramus. The necessity for accurate centring of the tube and correct positioning of the child is therefore obvious if a diagnosis is to be made at this stage. The delayed ossification of the femoral head may offer a clue but although ossification normally commences between the third and sixth month it must be remembered that all ossification dates are liable to considerable variations within the limits of the normal and delayed ossification is by no means uncommon in a perfectly normal hip. In unilateral cases however the difference between the two sides may be very helpful. Therefore while the diagnosis in a well developed case is perfectly obvious it is necessary to realise that cases of congenital dislocation cannot always be recognised with certainty in a radiogram at an early stage of the development of the condition.

During the course of treatment of a case of congenital dislocation the radiologist is called upon to decide whether the dislocation has been reduced or



not : in this connection it is important to be on the watch for fractures in the upper extremity of the femur, and since the limb will be in plaster, good radiograms are essential if fractures are to be recognised. They must be looked for especially in the case of patients who, having had their limbs in plaster for some time with failure to reduce the dislocation, are subjected to a further attempt at reduction before the bone has recovered from its period of disuse.

The results of treatment may vary considerably. Really good final results as judged radiographically are not as frequent as one might imagine, judging by the apparently successful reduction at the time of manipulation. The fail-



FIG. 270 Double congenital dislocation at the hip joint with the development of a false acetabulum in the ilium on each side

ure of the acetabulum to develop normally may defeat all attempts to retain the femoral head in the normal position for although in some cases after early reduction of the dislocation the acetabulum appears to develop normally, this is by no means the rule. Furthermore, the femoral head may be subjected to such a degree of trauma in the course of manipulation that its growth is impaired, and in this way again the hip joint does not develop normally, with the result that in old-standing cases it may be difficult to decide whether the condition is one of old osteochondritis of the femoral head or an old congenital dislocation which has been reduced.

#### Congenital Dislocation at the Knee Joint

A congenital dislocation at the knee joint usually shows forward displacement of the tibia on the femur. Clinically, these cases show the appearance of hyperextension at the knee joint. In genu recurvatum hyperextension is

also present due to laxity of the ligaments but in this condition the tibia is placed in normal relationship to the femur. If a genu recurvatum is untreated however a dislocation may develop. Congenital absence of the patella may occur in a case of congenital dislocation at the knee joint. Congenital dislocation of the patella is a rare condition. The patella is usually displaced outwards and may be associated with a flattening of the external condyle of the femur.

### **Congenital Dislocation at the Ankle Joint**

This usually occurs in association with bone defects. *Vollmann's congenital ankle deformity* is a condition in which the foot is developed normally but the fibula is rudimentary. The ankle joint is obliquely placed and the foot is in the valgus position.

## PART THREE

### SECTION IV

## TRAUMATIC LESIONS OF BONES AND JOINTS

BY

F. CAMPBELL GOLDING, M.B., M.R.C.P., D.M.R.E.

## FRACTURES OF THE FACIAL BONES

BY

H. K. GRAHAM HOGSON, C.V.O., M.B., F.R.C.P., F.F.R., D.M.R.E.

### CHAPTER XXIV

#### FRACTURES AND DISLOCATIONS

#### GENERAL PRINCIPLES

THE FUNDAMENTAL principle of securing radiograms in two planes has been established so firmly that it scarcely needs repetition. For many injuries an oblique projection is also desirable, to avoid a dense bone overlying a comparatively thin structure or to avoid superimposition. The experienced radiologist seldom has to resort to stereoscopic films in fracture work.

**The Recognition of a Fracture.**—Most fractures require little experience in interpretation, but all the information which is provided by the film is often not used. For example, it is important to realise that fissured fractures often involve the joint surfaces, and to note the presence of subluxation of the joint or some abnormality in the structure of the bone.

Pathological fractures may be suggested by the nature of the injury and confirmed by the radiogram. The bone in the region of a pathological fracture is usually altered in structure either by condensation or rarefaction, and the fracture line is frequently transverse. The radiologist examines with caution any fracture line which is exactly transverse.

**Mistakes in Diagnosis.**—Apart from the failures due to defective technique and poor radiograms, a fracture may be missed if the radiologist is unfamiliar with the lesions which are peculiar to special regions. Other errors are produced by the overlapping shadows of bones and soft tissues, by nutrient foramina in the bones, or by congenital abnormalities and epiphyseal lines.

**Union of Fractures**—The rapidity and security of union depend upon a number of factors among which are the site of injury the age of the patient and the presence of general or local disease

The presence of callus may be detected in favourable circumstances about the tenth day but fractures in some regions never show any callus and the presence of union can only be determined by the appearance of the cancellous structure across the fracture line

It is probably true that the radiologist waits too long before reporting firm bony union as judged by clinical standards Many fractures will bear the body weight without difficulty long before the film shows that union is complete

Union of fractures is sometimes delayed Some estimate of the prognosis may be obtained from an inspection of the surfaces of the fracture The prognosis deteriorates if there is evidence of bone absorption or sclerosis The sclerosis may appear as a thin layer of increased density which spreads across the opposing surfaces and then indicates the possibility of a pseudarthro is in other cases the sclerosis extends comparatively deeply under these edges especially in the presence of sepsis

### THE SKULL<sup>1</sup>

In infants part of the skull may be depressed without fracture In adults a fracture may be complete or incomplete the latter involving only one of the tables The inner table is usually affected as the resistance of the skull to pressure is greater than the tensile resistance a blow on the skull may therefore cause tearing apart of the inner table while the outer table returns to its normal position

A fracture may involve the vault or base of the skull or both the vault and base The type of fracture may be described as linear fissured comminuted depressed or compound with or without loss of bone

To obtain satisfactory films a standard technique is essential and stereoscopic films may be necessary In certain areas especially in basal fractures more information can be gained from a modified sinus and mastoid technique than from standard stereoscopic views It is essential to use fixed bony landmarks such as the orbito meatal line otherwise mistakes will be made in interpretation as a result of an unfamiliar projection

An immediate attempt to demonstrate the site and extent of the lesion is often difficult or undesirable it is also not necessary as fracture lines remain clearly defined for some time after the injury An early examination may be required to demonstrate a depressed fracture The depressed area is visible in a tangential view or the double density of the overlapping fragments will show in the ordinary film

<sup>1</sup> Fractures of the skull are further described particularly in the neurological aspects in the section on the Central Nervous System

Early recognition of the site of the fracture is also of value as it will indicate the possibility of complications such as hæmorrhage from a venous sinus or meningeal artery meningitis from a compound fracture opening into an air filled sinus or pneumatocœle capitis

Fractures of the vault frequently run downwards and forwards but they may travel in any direction or extend to the base. They occur usually in the line of the applied force and their direction is influenced to some extent by the weaker areas in the vault and base

The base of the skull is liable to fracture as parts are comparatively thin and there are numerous foramina which weaken the structure. The fractures are produced by direct or indirect trauma with irradiation or bursting. One or more of the fossæ may be involved. An annular fracture round the foramen magnum occasionally occurs due to an impact transmitted through the vertebræ

**Differential Diagnosis**—There are many shadows in a normal skull which simulate fractures. These are produced by sutures arteries and veins

A fracture line may vary in width or is widest at the middle the edges are very sharply defined it crosses arterial and other lines and tends to change direction by angles

*Sutures* appear as fine or dentate lines in constant positions. If a standard technique is always used the position of these sutures is soon recognised. The presence of congenital abnormalities should also be borne in mind such as non union of the two parts of the frontal bone forming the metopic suture. Another type of error is failure to recognise the line of non union between the basi occiput and sphenoid. In the adult the squamosal suture when seen in the antero posterior projection may be interpreted as a fracture. A suture may be widened by trauma but suture lines vary considerably in width in different individuals and the presence of diastasis is best determined by comparison with the opposite side of the skull

*Arterial grooves* are produced by the vessels of the meninges their edges are smooth and the grooves branch in arboreal fashion with progressive diminution in calibre

*The venous channels* are broad and their course is irregular. They may begin in small lakes in the bone and anastomose with other veins. The parietal vessels are often accentuated and tend towards a stellate form

## THE CLAVICLE

**Fractures of the Shaft**—Fractures of the inner half of the clavicle are rare. They are usually due to a severe and direct trauma. The most frequent site of injury is near the midpoint slightly towards the acromial end

*In infants* the greenstick fracture in this region is most difficult to detect and may be overlooked. This error is possibly due to the slight curve of the bone obscuring the fracture. Most radiologists have had the unfortunate

experience of having a fracture of this type returned with callus at the site, after they have given a negative report. This mistake can only be avoided by taking oblique views, a procedure which is hardly justified in routine work.

*In adults*, the fracture may be associated with angulation directed upwards and a dropped shoulder. Usually, however, there is gross displacement the outer fragment moving downwards, forwards, and inwards, producing some overlap. The displacement of the inner part is usually upwards and backwards.

Non union of a fracture is a rare event. The complete correction of the displacement is also rare. Union is nearly always associated with a radiological deformity.

At the acromial end of the clavicle, the fracture is transverse or oblique. The oblique fracture is more common on the lateral side of the trapezoid ligament. The joint may be involved and these lesions may simulate, or be associated with, acromio clavicular dislocation.

**Dislocation of the Acromio-clavicular Joint**—After injuries in this region the film will frequently show widening of the joint space on the affected side, presumably associated with an effusion into the joint. In frank dislocation the clavicle is raised above the acromion, and to demonstrate this it is preferable to examine the patient in the upright position with the arm hanging by the side.

One of the sequelæ of this injury is deposition of calcium in soft tissue. At the Royal National Orthopædic Hospital there were four instances of this in the year 1937. In each patient the heterotopic calcium and bone were visible in the space between the scapula and clavicle, apparently in the coraco clavicular ligament.

**Dislocation of the Sterno-clavicular Joint** is very rare. The direction of the dislocation may be upwards and forwards, or backwards. Satisfactory radiograms of this region are difficult to secure. The clinical examination is sufficient for the diagnosis.

## THE SCAPULA

Fractures of the scapula are rare. They may be classed as fractures of the body, spine, neck, glenoid cavity, coracoid and acromial processes. All fractures will not be demonstrated clearly in a standard film of the shoulder. In many cases modification of radiographic technique is desirable.

*The body of the scapula* is thin, and fracture lines may be difficult to detect. There is often comminution with overlap, and in this case the double density of the superimposed fragments will be visible (Fig. 280).

*Fracture of the spine* is usually associated with fracture of the body. Dislocation of the acromio clavicular joint is more common than fracture of the acromion process.

*Fracture of the glenoid cavity* is very rare, and usually involves the lower part of the joint surface

The various epiphyses, especially those of the acromial and coracoid processes and inferior angle of the body, should not be mistaken for fractures

## THE HUMERUS

**Upper Part**—*Displacement of the epiphysis and fracture of the anatomical neck* are rare. In some patients the fracture is in the region of the anatomical neck but not strictly confined to it throughout its length

Watson Jones has suggested a simple classification for injuries of the upper part of the shaft of the humerus as the customary subdivision into fracture of the anatomical and surgical neck has little to recommend it

(1) *Contusion crack fracture* due to a direct injury. The greater tuberosity is often comminuted. The crack in the neck is subperiosteal and may be difficult to detect

(2) *Adduction fracture*, equally common in adults and children. The fracture line may be oblique. The lower fragment is adducted with outward angulation, the greater tuberosity is intact

(3) *Abduction fracture* common in adults, uncommon in children. The shaft is abducted with inward angulation. The outer aspect of the neck receives the major thrust from the impact and the greater tuberosity may split off the bone

A pathological fracture through a cyst is not uncommon. The epiphyseal line has been mistaken for a fracture

In many injuries stereoscopic or lateral views are useful. The lateral radiogram taken through the chest is often very useful, and may reveal a marked displacement which was not visible in the antero-posterior film

*Fracture of the greater tuberosity* is one of the commonest injuries in this region. It may occur alone or be associated with fracture of the shaft (especially the abduction fracture), or dislocation of the joint (especially anterior dislocation). The amount of bone detached and the degree of dis-



FIG 280.—Subluxation of the acromio-clavicular joint. fracture of the acromion process, fracture of the base of the coracoid process, and fracture of the body of the scapula

placement vary considerably. The displacement is caused by the pull of the supraspinatus muscle.

Short of a frank fracture of the tuberosity, a common condition which is frequently overlooked is an elevation of a very small fragment of cortical bone at the insertion of various tendons in this region. The tendon most frequently responsible is that of the supraspinatus and more rarely the infraspinatus and subscapularis muscles. A lesser degree of trauma may produce only an irregularity of the outline of the cortex at the site of insertion of these tendons. In order to show the full amount of this injury, it may be necessary to take films with some rotation of the humeral head.

### Dislocation of the Shoulder Joint

Dislocations may be classified as

Forwards Subcoracoid → Subclavicular

Downwards Subglenoid

Backwards Subacromial → Subspinous

The subcoracoid dislocation is most common.

Other rarer types of lesion are luxatio erecta in which the arm lies vertically upwards and the head is subglenoid and subluxation downwards from weakness or palsy of the muscle especially the deltoid.



FIG. 81. Fracture of the caputellum of ten years duration.

Clinically there was cubitus valgus of 40 degrees.

One of the sequelae of this injury is late involvement of the ulnar nerve. This was not affected in this patient.

As complications of these injuries there may occur fractures of the greater tuberosity, neck of the humerus, glenoid and rarely the lesser tuberosity.

**Lower Part**—A supracondylar fracture is a very common injury in childhood. The fracture line is frequently oblique and directed upward and backwards with posterior displacement of the lower fragment which may be drawn upwards by the triceps (*extension fracture*). In some patients the fracture line runs in the reverse direction and the fragment is displaced upwards and forwards (*flexion fracture*). Either condylar surface may be fractured, more commonly the external. The detached fragment is often rotated (Fig. 281).

Another common injury is dislocation of the epiphyses of the epicondyles, particularly the internal epicondyle (Fig. 282). The latter may be detached by the tension of the flexor muscles, or by direct violence. Another explanation of the condition is that



there is a momentary subluxation of the joint with avulsion of the epiphysis by the internal lateral (ulnar collateral) ligament. The epiphysis may form a loose body in the joint.

In children the extension fracture is more frequently seen than the flexion fracture. In adults the condition is the reverse and the lower fragment is often T or Y shaped with or without separation of the fragments. Injuries in this region often unite with abundant callus which extends upwards along the shaft.



FIG. 8.—Avulsion of the epiphysis of the internal condyle.

### THE RADIUS AND ULNA

**Upper Part of the Radius**—The most common injury is a vertical crack in the head of the radius. It is important to realise that this lesion may be missed in films of poor quality. A sector of the head is often depressed and careful scrutiny of the cortex may also reveal a break on the under and outer margin of the head with slight overlap (Fig. 283). In some instances the whole head is comminuted.

Fracture of the neck is not so common as fracture of the head. When displacement occurs the lower fragment usually lies posteriorly; the head may or may not be held in position by the orbicular ligament.

**Lower Part of the Radius**—Fractures are liable to occur in the lower third of the radius. The accompanying displacement is influenced to some extent by the insertion of the pronator teres muscle. Above the insertion the upper fragment tends to be flexed and supinated by the biceps and supinated by the supinator brevis. Below the insertion the supination is neutralised to some extent by the pronator teres. In both instances there may be narrowing of



FIG. 23.—Fracture of a segment of the head of the radius.

the interosseous space due to the action of the pronator quadratus.

In children the lower third is the most frequent site of the 'greenstick' or 'bamboo' fracture of the shaft.

**Colles' Fracture**—A comparatively well defined group of fractures occur near the wrist joint. Several of these have received separate names or the whole group are classed as Colles' fractures.

The Colles' fracture occurs through the lower end of the radius at a varying distance from the articular surface, usually about half an inch. It may be transverse, fissured or comminuted. The detached fragment is displaced

upwards and backwards with impaction and with backward tilting of the radial articular surface. Lissures frequently involve the joint and are visible at right angles to the main fracture line. The bones of the distal radio ulnar joint may be separated and the styloid process of the ulnar is detached in over 60 per cent of patients. In the lateral view it is important to note the amount of dorsal tilt of the radial articular surface and this may be used as a measure of the success of attempts at reduction. In the postero anterior view the articular surface of the radius forms an angle of 30 degrees with the shaft and this should be restored.

Some variations of this lesion are

Fracture of the radius about 1 inch above the articular surface with the lower fragment completely detached and lying on the dorsum of the shaft of the radius

Fracture of only the dorsal half of the articular surface of the radius

Fracture of the radial styloid process

A fracture in an adult extending across the bone in a position which would correspond with the epiphyseal line. The lesion is characterised by an absence of displacement.

The reversed Colles fracture

In children the lesion which corresponds to the Colles fracture is a dorsal displacement of the radial epiphysis



FIG. 284.—Posterior dislocation of the elbow joint with fracture of the head of the radius and a fracture of the olecranon which is displaced upward.

The Ulna.—The most common injury in the upper part is fracture of the *olecranon process*. It is usually transverse but may be oblique when it approaches the coronoid process. The amount of displacement varies; the periosteum may be torn and the fragment elevated by the triceps.

The coronoid process is rarely detached. The line of fracture is usually parallel to the shaft and the fragment may be displaced upwards by the action of the brachialis anticus. The injury is often associated with posterior dislocation of the joint.

Dislocation of the Elbow Joint.—Such dislocations may be posterior (frequently posterior and slightly lateral) lateral anterior or medial or there may be a divergent dislocation of radius and ulna. Only the posterior (and lateral) dislocation is at all

common. It may be accompanied by a crushing injury of the head of the radius (Fig. 284).

Dislocation of a single bone is rare apart from forward dislocation of the head of the radius which is frequently incomplete, or associated with a flexion fracture of the ulna.

Dislocation of the head of the radius is an inevitable consequence of fracture of the ulna with overlap. The same principle applies to any of the paired bones. If one is fractured with appreciable overlap, then the other bone must fracture not necessarily at the same level, or one of the joints must dislocate. A congenital dislocation of the head of the radius is a rare finding.

## THE WRIST

**Fractures of the Carpal Bones**—A fracture may be found in any of the small bones of the wrist. Some of these fractures are rare. The proximal row are involved more often than the distal row and of these the number of fractures of the scaphoid is greater than the total of all other fractures of the carpus.

This bone is usually broken at or near the midpoint, but an avulsion fracture of the tubercle produced by tension on the radial collateral ligament and a fracture of the proximal end also occur (Fig. 285). Fractures of the tubercle always unite by bone.

The bone usually has two or more separate nutrient vessels one of which may be torn at the time of injury. In some patients, the vessel to the proximal half of the bone is absent and the fracture temporarily cuts off the blood supply. Any disturbance of nutrition tends to cause condensation or partial necrosis of the part involved.

Fractures of the scaphoid are easily missed and it is necessary to be able to distinguish between an old and a recent injury. In a recent fracture the edges of the fracture line are sharply defined and the cancellous tissue beneath the fracture retains the normal trabeculated appearance. After a few weeks some cystic degeneration or absorption of the opposing edges takes place and the appearance of the cancellous bone in the immediate neighbourhood of the fracture becomes blurred. At a later stage the opposing edges or one half of the bone may become sclerosed (Figs. 286 and 287). This appearance is accentuated by the osteoporosis of disuse in the remaining bones.

In the assessment of the amount of union after fracture it is necessary to



FIG. 285.—Fracture of the proximal part of the scaphoid. Fracture of the cuneiform. Fracture of the dorsum of the radial articular surface.

Dislocation of the semilunar. Same patient as Fig. 284.



FIG 286—Fracture of two years duration. No evidence of union. There are ill defined cystic areas beneath the fracture line. There is also some evidence of sclerosis of the bone particularly the proximal fragment.

Two types of *fracture of the semilunar* are described—fracture of one of the angles of the bone and a crush fracture. Fracture of a small particle at the posterior angle is comparatively common. This may occur alone or be associated with dislocation of the bone. The crush fracture is a very rare injury and may easily be confused with a post traumatic necrosis (*Kienbock's disease*).

Fractures of the remainder of the carpal bones are without particular features. Probably the most common is an avulsion fracture of the dorsum of the cuneiform. The particle detached is small and can be seen only in the lateral view.

**Dislocation of the Wrist**—Complete transverse dislocations may occur through the radio carpal, the mid carpal or the carpo metacarpal joints but all these types are rare. In the more common injuries the semilunar plays an essential part. Many of these dislocations are accompanied by fractures.

The most frequent injury is a *dorsal dislocation of the carpus on the semilunar and radius*. The subsequent behaviour

recognise that little callus is visible and reliance has to be placed on the appearance of the cancellous bone and on the continuity of this cancellous structure. In cases of non union a pseudarthrosis tends to develop with some sclerosis of the opposing surfaces. A possible fallacy in the X ray interpretation is failure to recognise a bipartite scaphoid. This finding is very rare and the diagnosis may be made on the fact that each part is surrounded by a thin but clearly defined cortex. The two bones are in fact separate ossicles. In addition congenital variations in development tend to be symmetrical and some assistance may be obtained from a radiogram of the opposite wrist.

In all cases of suspected injury to the scaphoid an oblique view of the wrist must be secured. The postero anterior and lateral films are not sufficient. By centring over the dorsum of the scaphoid with the radial border of the palm raised the bone is not foreshortened and even fine cracks will be visible.



FIG 287—The late result of a fractured scaphoid with non union and a dislocated semilunar. The semilunar and proximal fragment of the scaphoid show evidence of disturbed nutrition.

of the semilunar depends upon the state of the dorsal and volar ligaments. If these remain intact the semilunar maintains its normal relationship to the radius. The dorsal ligament is frequently torn (Fig. 288) and the semilunar is then free to rotate forwards and rotate on its own axis. If the semilunar rotates beyond 90 degrees the os magnum becomes opposed to the radial articular surface. The bony injuries which may accompany this displacement are fractures of the semilunar styloid processes of the radius and ulna or fracture of the scaphoid.



FIG. 288. Dislocation of the semilunar. Same patient as Fig. 285.

### THE FEMUR

Fractures of the Femoral Neck are common in females and in old age. The exciting cause may be an



FIG. 289.—A radiogram of an adduction fracture taken by portable apparatus. The patient is seated on a chair.

The position appears comparatively good but the lateral film showed that there was marked forward angulation of the proximal fragment. There was no impaction.

apparently trivial injury and the patient may not be referred to the radiologist at once. It is necessary therefore to be able to distinguish between an old and a recent injury and to bear in mind that pathological fractures occur at this site.

The capsule of the joint has an oblique attachment to the neck and the classical grouping of intra and extracapsular fractures is unsuitable in radiological diagnosis. These fractures may be described as—(1) subcapital (2) mid-cervical (3) fracture of the base of the neck and (4) intertrochanteric.

There are two types of subcapital fracture. The most common injury is the adduction fracture the characteristic features of which are—coxa vara, absence of impaction, rotation of the head so that it appears below the acetabulum, outward rotation of the leg, with increased prominence of the lesser trochanter, forward angulation in the lateral view (Fig. 289).

The abduction fracture is comparatively rare. The radiological features

are coxa valga impaction head rotated so that part appears beyond the lateral margin of the acetabulum inward rotation of the leg with decreased prominence of the lesser trochanter The fracture line may be difficult to detect Bony union always takes place

In *subcapital fractures* the lesion may be with or without displacement and is rarely impacted Union usually results in a varus deformity In fractures in this region union appears to be more probable the nearer the fracture approaches the trochanters



Fig. 290 An intertrochanteric fracture

*Fractures of the base of the neck* are usually impacted the upper fragment being driven for some distance into the shaft The impaction is greater at the inner margin and so the position is one of varus The normal angle of the neck is approximately  $120^\circ$  degrees

In *intertrochanteric fractures* the fracture line is oblique corresponding more or less closely with the anterior oblique or intertrochanteric line The fracture line may be more horizontal the major part of the greater trochanter remaining attached to the proximal

fragment The lesser trochanter is frequently detached in these injuries and displaced upwards by the ilio psoas muscle (Fig. 290)

*Union of a fractured neck* may be bony or fibrous and delayed or absent Intertrochanteric fractures always unite by bone Above this region it may be difficult to determine from the film if satisfactory union has been attained and fibrous union cannot be detected In most patients little or no callus forms In others it is possible to tell that union has not and will not take place In these individuals some blurring of the structure of cancellous bone is visible at the margins of the fracture followed by a rapid absorption of the bone Almost the whole of the neck may be absorbed (Fig. 291)

**The Shaft of the Femur**—The radiological diagnosis of fractures of the femoral shaft as of other long bones does not usually present any particular difficulty The greatest use of X rays lies in the assessment of the position of

the injury, the amount of comminution, the result of reduction or traction, and finally the degree of union. A common radiological fallacy is the confusion of oblique with spiral fractures. This is more likely to occur when one fragment is rotated.

Apart from the direct influence of the trauma, there are certain characteristic deformities associated with the level of the lesion. In the upper third, the proximal fragment is flexed by the iliopsoas and rotated outwards and abducted by the muscles inserted into the greater trochanter, the lower fragment is displaced upwards and medially. In the lower third, the gastrocnemius



FIG. 291.—Old fracture, with non union and absorption of the neck. The head has undergone some sclerosis from defective blood supply.

muscle tilts the lower fragment backwards, and the upper fragment tends to approach the suprapatellar extension of the joint or may be adducted.

#### Dislocation of the Hip Joint.—

The hip may be dislocated backwards or forwards :

*Posterior* : Sciatic, iliac.

*Anterior* : Obturator, pubic.

The iliac and sciatic dislocations are most common.

Some difficulty may be experienced in distinguishing between the radiological appearances of traumatic, pathological, and congenital dislocations.<sup>1</sup> The pathological dislocation may be detected by the history and the presence of irregularity of the bone due to the disease process. In the very early stage,

<sup>1</sup> Congenital dislocation of the hip joint is further considered in the section on Congenital Deformities of Bones and Joints.



FIG. 292.—A comminuted and impacted "T" shaped fracture of the tibia, and fracture of the upper end of the fibula. The position has been partially corrected, and the radiogram was taken through the plaster.



FIG 293 Depressed fracture of the tibial articular surface due to the impact of the bumper bar of a car

before destruction is visible the history alone is sufficient

The early congenital dislocation is associated with

(1) Retarded epiphyseal development

(2) A sloping roof of the acetabulum

(3) Delay in bony union of the pubis and ischium with a wider gap on the affected side

In the later stages some aplasia of the limb is evident and a neoarthrosis forms above the old acetabulum which tends to be obliterated. The inlet of the pelvis is more rounded on the affected side. The femoral head is always deformed.

### THE KNEE JOINT REGION

**The Lower End of the Femur and Head of the Tibia**—There is a certain similarity in the radiological appearance of condylar fractures whether of femur or tibia. In either case there may be

(1) An oblique or comminuted fracture of a single condyle or part of a condyle

(2) A T or Y shaped fracture with some separation of the fragments by the end of the shaft (Fig 292)

One of the commonest injuries is a fracture of the external condyle of the tibia part of the articular surface being depressed below the remainder (Fig 293). The head of the fibula is often fractured in this type of injury.

Fracture of the internal condylar surface is comparatively rare. In either injury a valgus or varus deformity is usually present.

Fracture of the intercondylar eminence is of some radiological importance as it may be overlooked



FIG 294 Valgus on of the tibial plateau leaving a saucer shaped depression



The fracture is caused by strain on the cruciate ligaments. The anterior cruciate ligament which is usually involved is inserted into the internal spine and anterior part of the intercondylar fossa. Elevation of this fragment therefore fractures the internal spine and leaves a saucer-shaped depression on the anterior part of the tibial surface in the lateral view (Fig 294).

**Fractures of the Patella**—These are produced by indirect and direct violence. The most common injury is a transverse crack resulting from the contraction of the quadriceps over a flexed knee. A more severe strain tears across the soft tissues and produces a varying degree of displacement and rotation of the fragments. A direct blow on the patella tends to produce fissured stellate or comminuted fractures usually without much displacement.

It is necessary to be able to recognise a patella that has developed from two or more centres which have not fused. This may be done by noting the smooth dense edges of the parts and by the tendency to symmetry of congenital abnormalities. It should be noted that fracture of the patella is practically never found in children or adolescents.



FIG 293—An axial view of the patella. The normal appearance appears to have been fractured but the lucency is a congenital abnormality.



FIG 294—An injury due to forcible abduction of the foot resulting in a greenstick fracture of the fibula and partial dislocation of the tibia.

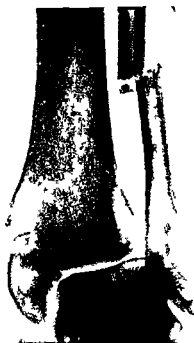
The fracture may be missed if sufficient penetration is not used to show the patella in the ordinary films and in some cases oblique views may be useful (Fig 295).

**Fracture of Upper Part of the Fibula**—This may be found as a single lesion. It may also be associated with fracture of a tibial condyle or fracture of the lower third of this bone. The only precaution which should be taken in diagnosis is to use a film of sufficient length to cover this part of the shaft especially in displaced fractures of the lower part of the tibia.

**Dislocation of the Knee Joint**—The joint may be dislocated forwards backwards laterally or medially. The radiologist seldom sees these conditions.

It is important to recognise minor displacements due to rupture of the cruciate and collateral ligaments by the abnormal width of the joint space on one side.

*Lateral dislocation of the patella* is prone to occur in patients with genu valgum. The patella may also rotate about its long axis to the extent of



90 degrees or 180 degrees Upward or downward dislocation may follow rupture of the patellar ligament or tendon of the quadriceps muscle

### THE ANKLE AND FOOT

**Fracture dislocations of the Ankle Joint**—Fracture of the fibula in the lower third may occur alone or be associated with an extensive injury to the ankle joint

Fractures in this region often termed loosely as Pott's fracture may be classified as

- (1) Rotation fractures
- (2) Abduction fractures
- (3) Adduction fractures
- (4) Fractures with posterior dislocation
- (5) Fractures with anterior dislocation

The rotation fractures are produced by forcible external rotation of the

**FIG. 97** The adduction fracture. The astragalus has been subluxated with tearing of the interosseous ligaments. Both margins of the tibial articular surface have been fractured.

foot this is usually brought about by internal rotation of the trunk the foot being fixed on the ground. In its simplest form it results in an oblique fracture of the fibula the fracture line running upwards and backwards. In a more severe injury the astragalus is dislocated outwards backwards and upwards the fibula and posterior surface of the tibia are fractured and the internal malleolus avulsed.

In the abduction fractures the fibula breaks transversely about the level of the ankle joint or the fracture may be considerably higher. The tibiofibular joint may be widened and the internal malleolus may also be fractured trans-



**FIG. 98** The adduction fracture. The vertical direction of the fracture line in the tibia characteristic. The top of the fibula has also been fractured.

versely with some lateral displacement of the astragalus (Figs 296 and 297)

In the adduction fractures the direction of the applied force is reversed. The fibula usually breaks at the tip or at the level of the joint and the fracture line in the internal malleolus is nearly vertical (Fig 298)

A patient who is knocked down by a blow on the side may therefore receive an adduction fracture of one ankle and an abduction fracture of the other

The posterior fracture dislocation is not common and the anterior fracture-dislocation is very rare. In each case the astragalus is displaced and a triangular fragment of the tibia is detached with a vertical fracture line

The Astragalus.—The head, neck, body, and posterior process may be involved. Dislocation is a common accompaniment of the major injuries. Fracture of the posterior process may present some difficulty as there is a close resemblance to the normal os trigonum. The diagnosis may be aided by comparison with the opposite side and by the presence of the fresh fracture surfaces



FIG 300.—The lateral view of a fracture of the os calcis. The condition had been present two and a half years and was lateral. (The patient had tried to come to the side by jumping from a height.)

An attempt to produce a sustentaculum tali arthrodesis has failed

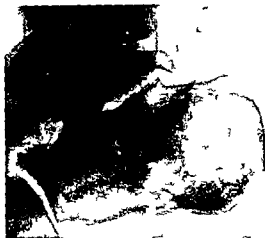


FIG 299.—A recent crush fracture with a reduction in the tuberosity joint angle.

Os Calcis.—The recognition of a fracture of the os calcis is most important. Clinically the condition may easily be confused with a sprain or a malleolar fracture. The diagnosis may be difficult in films of good quality and impossible in poor films. The examination should always be made in two planes

Several rare types of injury have been described such as fracture of the sustentaculum tali, peroneal tubercle avulsion fracture of the tuberosity, or fracture of the upper and medial parts of the tuberosity

The crush fracture is the most common injury (Fig 299). There may

be no displacement, and the fracture line may only be recognised by a slight alteration in the appearance of the cancellous bone or by the presence of a fine crack in the cortex. *Bohler* has described a method by which the amount of deformity may be gauged. Two lines are drawn along the upper surfaces of the bone, from the anterior and posterior angles to the highest point on the bone. These usually meet at an angle of 140 degrees to 160 degrees, the complementary angle is 20 degrees to 40 degrees, and is called the tuberosity-joint angle. Any fracture with impaction reduces this angle (Fig. 300).

Displacement may involve the posterior part or the whole of the articular surface with the astragalus. A crushing injury of the anterior part of the bone similarly disorganises the calcaneo cuboid joint, or the midtarsal joint may be subluxated.

**The Scaphoid.**—Two types of fracture occur, a crush fracture of the body and an avulsion fracture of the tubercle. In the former the lower part of the bone is crushed and the upper part displaced upwards. The astragalo scaphoid ossicle and the os tibiale externum should not be mistaken for fractures.

**The Metatarsals.**—Fractures of the metatarsals may be due to direct or indirect injury. The former are frequently multiple.

Fracture of the proximal end of the shaft is easily overlooked, if the part is not examined carefully.

Indirect injury is responsible for a fracture of the base of the fifth metatarsal (the *Robert Jones* fracture), and the march fracture. The epiphysis for the tuberosity of the fifth metatarsal should not be mistaken for the former.

The march fracture has been described in the second, third, and fourth metatarsals, but only the second metatarsal is commonly involved. The predisposing condition is prolonged strain (e.g. of marching) in the presence of a defective bony architecture. The structural faults have been described as (1) a short first metatarsal, (2) sesamoids of the great toe placed more proximally than usual, and (3) hypermobility of the joint between the first and second cuneiforms (*Morton*).

Any of these factors tends to throw excessive weight on the second metatarsal, resulting in a spontaneous fracture. The fracture line is difficult or impossible to detect, and attention is frequently directed to the condition by the presence of periostitis or callus. The callus is often excessive, due to lack of treatment. The condition has been mistaken for an osteogenic sarcoma.

### THE PELVIS

Pelvic fractures are frequently multiple and a certain amount of care is necessary in diagnosis, otherwise one of the lesions will be missed.

A double fracture into the obturator foramen is common, the upper fracture is sometimes oblique and more difficult to detect than the lower. Fractures in this region may also be associated with separation of the symphysis pubis, or

dislocation of the sacro iliac joint and fracture of the posterior pelvic wall on the same side (*Malgaigne's fracture*).

Fractures of the iliac crest are frequently comminuted. The rim of the acetabulum is rarely involved in a dislocation of the hip. The os acetabuli and strip epiphysis for the margin of the acetabulum should not be mistaken for fractures. The floor of the acetabulum is sometimes fractured, and the head of the femur may be driven into the pelvis (central dislocation).

### THE RIBS

The impression is conveyed in many text-books of surgery and orthopaedic surgery that the radiological diagnosis of fractured ribs is unreliable. This statement is true, if the diagnosis is attempted on a single film of the chest. In addition to the postero-anterior film, it is essential to have two oblique views, otherwise the curvature of the rib will obscure the fracture line by the superimposition of intact bone. Fractures below the diaphragm will also be missed if sufficient penetration is not used.

The fracture line is oblique or transverse and heals by bony union, wide separation or displacement is uncommon. Confusing shadows are produced by overlapping bone and by the thoracic contents. The latter shadows will be found to continue beyond the rib substance into the intercostal spaces.

### THE SPINE

Plain antero-posterior and lateral films are usually sufficient to demonstrate the extent of a fracture, but certain regions may present some difficulty. In the cervical spine, the bodies and their appendages are closely packed, and stereoscopic films may give additional information, especially when the area is obscured by a dislocation (Fig. 301). In fractures of the upper cervical spine, an antero-posterior view should be taken through the open mouth. In broad and muscular individuals, the lower lumbar and upper four dorsal vertebrae are difficult to show satisfactorily in the lateral view, and oblique films of the latter may be helpful.

A fracture may involve the spinous or transverse process, the lamina, pedicle, facet, or body of the vertebra. The cause may be direct and indirect trauma or muscular action.



FIG. 301.—Dislocation of the cervical spine. Stereoscopic films showed a fracture of the intervertebral facets.

*The most common site of injury is from the twelfth dorsal to third lumbar vertebrae inclusive. The spine from the first to tenth dorsal vertebrae is less mobile and less liable to injury than the regions above or below. In a series of patients which has been collected in the Toronto General Hospital from 1907 to 1926 there were 34 cervical 10 dorsal and 88 dorso lumbar fractures. In this series the last two dorsal vertebrae were included in the lumbar region.*

The proportion of patients with cord involvement is highest in fractures of the dorsal region and the number with multiple fractures is also relatively higher in this region.

**Fractures of the Laminæ** are very rare. They are due to direct violence and may occur in any region. They are frequently associated with cord injuries and careful inspection is necessary for their detection.



FIG 30 A recent crush fracture



FIG 303 The same patient as Fig 30, three months later. A line at on into the fractured body is now visible

**Fractures of the Spinous and Transverse Processes** due to direct injury are uncommon in the dorsal region as they are protected by the ribs and the spinous processes do not project to any extent. The spines of the seventh cervical and first dorsal vertebrae are relatively prominent and are not infrequently fractured often by muscular effort. Fractures of the transverse processes in the lumbar region are not uncommon and are frequently multiple with wide separation of the fragments. The third and fourth vertebrae are usually involved.

**Fractures of the Body**—Most vertebral fractures are due to the indirect trauma of crushing injuries with hyperflexion of the spine. The amount of deformity is proportional to the severity of the injury and in some patients only the slightest wedging of the vertebral body may be visible.

One vertebra only is usually involved. Multiple fractures do not necessarily

involve neighbouring bodies and there may be several intact vertebrae between two lesions.

The upper margin of the body is usually depressed to a greater extent than the lower<sup>1</sup> (Figs 302 and 303) and occasionally a wedge shaped fragment is detached from the antero superior border. This fragment may resemble the ring epiphysis in the lateral view and it may be most difficult to distinguish between a recent fracture and an ununited epiphysis.

As would be expected in a crushing injury the deformity in outline is the most prominent feature and fracture lines are obscured. Sometimes a break is apparent in the contour of one of the margins (Fig 304) or impaction with overlap can be detected on the anterior surface of the body. In other patients the body is reduced in stature especially at the anterior border with an increase in convexity of this surface and a compensatory increase in width of the whole body. The posterior surface of the body derives considerable support from the intervertebral joints and is less commonly involved. Additional features which may be noted on a film are minor degrees of subluxation of a body, dislocation or fracture of the intervertebral facets, projection of part of the body into the neural canal, narrowing of intervertebral foramina.



FIG. 304.—Fracture of the body of lumbar I due to a skiing accident.

In moderately severe injuries the disc space is preserved or may appear wider than normal in its anterior part. In severe injuries some of the nucleus pulposus may be driven into the body of the vertebra with some narrowing of the disc due to the loss of substance. In films of good quality these herniations may be visible especially after the lapse of some months.

As a late result a fracture may terminate in a wedged deformity of the body without any further evidence as to the cause of the condition. The fracture lines are invisible and the outline of the body is unbroken. The more severe injuries usually cause a wedged vertebra and ultimately gross localised osteoarthritic changes at the site (Fig 305).

A wedged deformity of one or rarely of two bodies may be discovered months after an accident which did not appear to have caused a fracture at the time.

<sup>1</sup> See also Vol III Part III Sect 6.

It has become a common custom to refer to this condition as Hummel's disease (92)

**Fracture of the Atlas** is atypical in appearance because of the difference in its architecture. The lesions may involve either the arch or the lateral mass and there may be more than one fracture.

Fracture of the posterior arch is most common. Fracture of the arches appears to be due to the tensile force exerted by the lateral masses which tend to separate owing to the obliquity of their articular surfaces.

The most common complications of fracture of the atlas are fracture of the odontoid process and dislocation with rotation.

**Differential Diagnosis** — The radiologist must frequently differentiate between fracture, tuberculosis and secondary neoplasm in the spine.

The typical features of *tuberculosis* are early loss of the disc space and erosion of some part of the body. The early formation of pus visible in the form of a paravertebral shadow or the psoas abscess may be a useful clue.

In *neoplasm* the disc space is usually preserved but there are marked changes in the body. The common deposits cause intense local destruction and rarefaction; the cancellous structure is destroyed with collapse of the body or the body tends to disappear. The lesions are often multiple.

Among the less common conditions which may have to be distinguished from fracture is the *malacia* produced by osteoporosis from any cause. It is probably most common in senility. Some of the bodies may be porous to such an extent that they collapse or the turgor of the discs indents them so that they resemble the vertebrae of a fish. A fracture may be superimposed on this condition. The *solitary dense vertebra* is usually due to osteoplastic secondaries. Paget's disease, Hodgkin's disease or disturbance of nutrition. It is unlikely that the condition will be confused with fracture.



FIG. 35.—The late result of a fracture healed occurred fourteen years previously.

#### REFERENCES

- NORTON DUDLEY J. *The Human Foot* Columbia Univ. Press 1930  
 WATSON JONES R. *Proc Roy Soc Med* 1935 XXIX 1638



## CHAPTER XXV

### LATE SEQUELÆ OF TRAUMA OF BONES AND JOINTS

#### SLIPPED EPIPHYSIS OR ADOLESCENT COXA VARA

IN THIS condition a varoid deformity develops at the hip joint due to slipping of the capital epiphysis of the femur. The onset occurs in adolescence.

**Incidence**—Males are affected more frequently than females in the ratio of 3:1. It has been described in patients between the ages of 9 and 19 years. The average age at the onset in females is approximately two years less than that in males. The left hip is affected more often than the right. In approximately 10 per cent. of patients the condition is bilateral.

**Ætiology**—Many theories have been evolved to explain this condition. In adolescence the epiphyseal line assumes a more vertical position and is probably the *locus minoris resistentiæ* (Key).

**THE TRAUMATIC THEORY**—In all the larger series of cases which have been reported this factor has been mentioned in some of the patients. In a number the condition has been found immediately after trauma although this is often not severe. In others it has been supposed that the epiphysis moves under the influence of continual slight trauma, none of which is sufficient to draw attention to the joint at the time.

**THE STATIC THEORY (Hofmeister)**—This assumes a disproportion between the weight of the individual and the strength of the neck in the rapidly growing patient. The protagonists of this theory do not admit the presence of any pathological change in the cartilage or bone.

**THE THEORY OF ASSOCIATED DISEASE**—At various times especially in the early literature patients have been reported in whom the authors have considered that the process was initiated by some disease. Among these conditions are

Obesity due to endocrine dysfunction (Kärmisson, Piculet, Ridlon, Hass, Riedel, Wilson, Willis, Wardle). Many patients are of this type.

Rickets (Fiorani, Muller, Keetley, Haedke, Bloch, Frösch, Brailsford).

Localised osteomalacia (Kocher).

Arthritis deformans (Monks and Clarpentier, Mayall).

Some disease of cartilage of unknown origin (Schwart).

An infection by low grade organisms (Froelich, Ferguson and Howorth).

A localised disturbance of blood-supply (Iselin).



FIG. 306 There is evidence of early slippage of the left capitol epiphysis as shown by the lack of definition of the extremity of the neck. Compare the outer contour of the head and neck on the two studies.



FIG. 307—B lateral slipped epiphyses



FIG. 308—A more marked deformity than in the previous patient with further changes in the metaphyses.



FIG. 309 The late result of a slipped epiphysis at the age of 13 years with a typical prominence at the junction of the head and neck on the surface.

Thinning of the periosteum of the neck associated with rapid growth  
(*Key Badgley*)

**Pathology**—In 1926 there were reports in the literature of twenty four patients (*Key*) in whom exploration had been performed with an examination of the specimen. Since this time other reports have appeared but the pathology of the condition is still obscure.

In the early stage there is a slight widening of the epiphyseal line with irregularity of the arrangement of cartilage cells and increased vascularity. The epiphyseal line becomes irregular with invasion by spongy bone. Some extravasation of blood occurs and the head is united to the neck by soft callus and fibrous tissue.

No uniform disease process has been reported in these sections.

**Radiographic Appearances**—The initial stage is indicated by a slight broadening of the epiphyseal line. This has been described as a 'preslipping' phenomenon but it is possible that the epiphysis has commenced to move by the time this is evident on the film. The juxta-epiphyseal bone of the head remains intact and clearly defined, whereas the bone on the opposite side of the epiphyseal line is altered in structure, the sharp margin of the neck is lost, and cancellous network of the extremity of the neck is poorly defined. In the early stage of slipping one of the most reliable signs is an alteration in the contour of the outer margin of the head and neck, comparison being made with the opposite side (Fig 306). In the affected hip the upper part of this curve becomes flatter. As slipping increases the changes in the proximal end of the neck become more marked. The head moves backwards and downwards with internal rotation with the result that a varying part of the lower half lies outside the acetabulum and is visible below the neck (Figs 307 and 308). The neck is therefore thrust forwards or may appear to be partly embedded in the head in a position of external rotation. Repair is effected by the formation of new bone which unites the head to the shaft in a position of varus (Fig 309). The epiphyseal line is obliterated earlier in the deformed hip. In the final stage *Kocher's* three postulates may be fulfilled. These are lengthening of the outer border, forward bowing and torsion of the neck. When union has taken place the outer and upper extremity of the neck frequently remains as a prominent boss of bone. Secondary effects of the deformity which may appear at a later date are modifications of the shape of the acetabulum, ischium, varus and osteoarthritic changes.

**Differential Diagnosis**—Clinically this condition is frequently mistaken for tuberculous but after a radiogram is taken no difficulty should arise as the osteoporosis and erosions of tuberculous do not resemble coxa vara.

In congenital or infantile coxa vara the age of the patient when brought for treatment is usually younger with a history of defective gait since walking. The condition may be bilateral with a characteristic vertical break in the neck which may enclose an inverted V shaped fragment of bone. The

greater trochanter is elevated and beaked. There are frequently other congenital abnormalities.

In *rachitic cora vara* the changes in the epiphyseal line are visible with other rachitic lesions in bones.

In *osteochondritis* of the hip the patient is usually under 10 years of age. The head remains in position but shows the increased density and islands of sclerosis and rarefaction associated with osteochondritis in any situation.

### THE PELLEGRINI STIEDA LESION

This abnormality develops one or two months after a knee strain or a direct blow on the internal epicondyle of the femur. It consists of a deposit of heterotopic bone which develops in the soft tissue in the region of this epicondyle and the adductor tubercle. After a prolonged period of rest the deposit tends to become smaller and more dense or small deposits may be entirely absorbed (Fig. 310).

Stieda believed that the lesion was a chip fracture. Cases have been seen in which there was both a chip fracture and this deposit of bone. The etiology of the condition appears to be the same as that of myositis ossificans (*q. v.*). The condition has been operated upon by Kulowski, Finner and Riebel, Riebel and Riebel and Collart; the deposit always recurred after operation. At operation the flake of bone was found to be covered with fibrous tissue and attached to the tibial collateral ligament. If the deposit was large the upper part extended anterior to the tendon of the adductor magnus or infiltrated the tendon of the adductor magnus and vastus medialis (Kulowski).



FIG. 31. A comparatively small area of calcification on which followed a football injury.



FIG. 311.—A heterotopic deposit of bone discovered four months after a blow on the knee.

A similar flake of bone which bears a striking resemblance to this lesion may be found after an injury on the curved surface of the posterior aspect of the lower end of the tibia (Fig. 311).

## KÜMMELL'S DISEASE

In 1891 *Kummell* described a condition of post traumatic rarefying osteitis of a vertebra which after a latent period gave rise to symptoms of vertebral collapse. The diagnosis was based on clinical evidence.

Over two hundred articles have appeared in the literature since this time, and the interpretation of 'Kummell's disease' has varied considerably. The necessary desiderata appear to be

- (1) An injury to the spine often not severe
- (2) A latent period of months or even years
- (3) Objective symptoms of collapse with a local kyphosis most frequently in the dorsal region with subjective symptoms of local pain girdle pain or cord involvement

To these should be added the radiological evidence of

- (4) Rarefaction
- (5) Absence of deformity immediately after the accident
- (6) Presence of deformity at a later date

*Glaser* and *Rigler* have found the condition in more than one vertebra.

*Kummell* believed that the bone was injured but not fractured and *Ludloff* described the post mortem findings of multiple small hematomata in a vertebral body after injury. *King* has observed also at post mortem excessive vascularity of the body which he believed accounted for the rarefaction during the latent period.

The radiological appearances are those of a simple wedged deformity often with some increase in the disc space. The demonstration of rarefaction in those articles which show prints before and after the injury is not impressive.

The probable explanation is that the condition is due to an unrecognised fracture. This theory appears more likely when comparison is made with the os calcis. This bone resembles a vertebra in structure and is subject to crushing injuries yet fractures which are not accompanied by impaction may be most difficult to detect in spite of its superficial situation and the fine detail which can be reproduced on a radiogram.

## POST-TRAUMATIC CYSTIC LESIONS

Small cysts may form in bone as the late result of a severe trauma or recurrent mild trauma.

They may be found as single cysts in the carpus



FIG 31.—Cystic changes in the head of the first metatarsal probably due to recurrent trauma associated with the abnormal prominence of the bone.

usually in the scaphoid and semilunar, or involving several bones (*Eaton*) especially in compressed air drill workers (*Brailsford*) Other sites which have been mentioned are the trapezium (*Kohler*) the os magnum and unciform (*Buchman*) and the ulnar styloid (*King*) They are occasionally seen on the inner surface of the head of the first metatarsal in hallux valgus (Fig 312) and have been reported in other rare sites by *Duncker* and others

The lesions are characterised clinically by an injury, a latent period and finally development of symptoms

## REFERENCES

*Adolescent Coxa Vara*

- BADGLEY C C *Journ Amer Med Assn* 1929 XCII 355  
 BLOCH R *Journ de Chir* 1921 XVIII 1  
 BRAILSFORD *Lancet* 1933 I 16  
 CHARPENTIER *Thèse de Paris* 1897 (quoted from Lauper)  
 FERGUSON A B and HOWORTH M B *Journ Amer Med Assn* 1931 XCII 18  
 FIORANI *Gazz d osp* 1881 *Abs Zentralbl f Chir* 1882 IX 265  
 FROELICH *Ztschr f Orthop Chir* 1904 XII 80  
 FROSCH E *Munch med Wchnschr* 1919 LXVI 375  
 HAEDKE M *Deutsche Ztschr f Chir* 1903 LXVI 89  
 HASS *Zentralbl f Chir* 1923 L 283  
 HOFMEISTER F *Coxa Vara Joachimstals Handbuch der Orth Chir Jena* 1905 p 362  
 IRWIN S T *Irish Journ Med Sci* 1929 VI 71  
 ISELIN H *Schweiz med Wchnschr* 1924 VI 157  
 KEETLEY A *Journ Med News* 1888 I No 7 quoted Whitman R V 1 M 1  
*Journ* 1894 LIX 769  
 KEY J A *Journ Bone and Joint Surg* 1926 VIII 53, *South Med Journ* 1924 XVII 959  
 KIRKISSON M E *Bull de l'Acad de Med* 1918 LXXIX 183  
 KOCHER T *Deutsche Ztschr f Chir*, 1894 LXXXVIII 521  
 LAUPER J *Deutsche Ztschr f Chir* 1912 CXXI 347  
 MAYDI *Abs Zentralbl f Chir* 1897 XXIV 117  
 MÜLLER E *Beitr z Klin Chir* 1889 IV 137  
 RIDLOX J *Journ Amer Med Assn* 1915 IXX 219  
 RIEDEL G *Zentralbl f Chir* 1923 L 312  
 SCHWARTZ E *Beitr z Klin Chir* 1913 LXXXVII 709  
 VIFULLET H *Presse Med* 1924 XXXII 537  
 WARDLE F V *Brit Journ Surg* 1933 XXI 313  
 WILLIS T A *Journ Bone and Joint Surg* 1929 XI 779  
 WILSON P D *Journ Amer Med Assn* 1924 LXXXIII 1749

*Pelligrini Stieda Lesion*

- COLTART W D *Proc Roy Soc Med* 1938 XXI 180  
 FINDER and RIEBEL *41st Journ Surg* 1934 CII 17  
 KULOWSKI J *Journ Amer Med Assn* 1934 C 1014  
 RIEBEL and RIFBEL *Amer Journ Surg* 1934 XXIV 181

*Kummell's Disease*

- GLASER F *Med Klin* 1918 XIV 360  
 KING E S J *Localised Rarefying Conditions of Bone* 323 (Edward Arnold London 1935)

- KÜMMELL, H, *Verhandl d 64 Vers deutsch Natur u Arzte* in Halle, 1891, S 282,  
*Deutsch Med Woch*, 1895, XXI 180, H, *Arch f klin Surg*, 1921, CXVIII, 876,  
*Monatsschr f Unfall*, 1928 XXV, 63, H, *Arch f Orthop*, 1928, XXVI, 471  
 LUDLOFF, K, *Fortschr a d Geb d R nt*, 1906, IX 175  
 RIGLER, L G, *Amer Journ Roent*, 1931, XXI, 749

*Post traumatic Cystic Lesions*

- BRILSFORD, J F, "The Radiology of Bones and Joints," London, 1930  
 BUCHMAN, J, *Ann Surg*, 1928, LXXXVII, 892  
 DUNCKER, *Fort a d Geb d R nt* 1928, XXXVIII, 1123  
 ESAU, P, *Zentralbl f Chir*, 1928 LV, 1803, *Deutsch Ztschr f Chir* 1931, CCXXXIV,  
 560  
 KING, ' *Localised Rarefying Conditions of Bone* London, 1935  
 KOHLER G D *Fort a d Geb d R nt*, 1926, XXXIV, 342

## CHAPTER XXVI

### FRACTURES OF THE FACIAL BONES

PRIOR to the 1914 War fractures of the facial bones were a comparatively unusual occurrence but with the vast increase in the general use of the motor car and especially since the advent of unbreakable glass they have become increasingly common.

In the days when ordinary glass was used in windscreens the victim of a motor smash frequently suffered severe cuts and lacerations from the flying glass but nowadays he comes in violent contact with a hard unyielding substance with resulting fractures. Although fractures affecting the facial bones are not usually dangerous to life they may cause varying degrees of disability by involving the orbit temporo mandibular joint etc but their main sequel if the extent of the injury is not recognised and treated before union has commenced to take place is permanent disfigurement. This may not affect the man in the street to any appreciable extent but to a young woman it may be a severe handicap and to a screen star it may be of paramount importance. If recognised in time these deformities can be corrected in such cases by surgical means but owing to the swelling of the soft parts a facial bone fracture may frequently escape notice until it is too late to correct it union having taken place. It is therefore of importance to be able to recognise this increasingly common type of fracture at the outset.

Not infrequently one sees old and united depressed fractures of the lower orbital margin producing obvious disfigurement but which have passed unrecognised for some time after the injury owing to the swelling of the soft parts.

The most certain method of recognising these fractures is by X rays but unless the X ray examination is carried out with a full knowledge of the anatomy of the parts and the mechanics of this type of fracture the bony injury may even then escape notice. In other words the radiologist has to take into account the direction of the force visualise the plane of the possible resulting fracture and take radiograms so as to view it tangentially and with the correct degree of X ray penetration to throw the particular bone or bones concerned into relief.

The stereotyped antero posterior and lateral radiograms of the skull will show one little or nothing of this type of injury and special views have to be taken with this particular object in view.



## THE NASAL BONES

The commonest of the facial bone fractures, and probably the most disfiguring, are those of the nasal bones. The deformity may be a severe depression of the nasal crest, producing irregularity of the profile of the nose (Fig 313). On the other hand, the lateral view (Fig. 314) may show simply a linear fracture,



FIG 313—Fracture of the nasal bones with depression of the fragments



FIG 314—Fracture of the nasal bones; no deformity is visible in this view (cf Fig 315)

with apparently no displacement, yet the axial view (Fig 315) may show a marked lateral displacement of the frontal process of the maxilla, the nasal bone, the septum, or of all three

It is obvious, therefore, that, in such cases, views must be taken in more than one plane, and these planes together with the exposures used, must be such as to throw the nasal bones and the frontal processes of the maxillæ into relief.

An ordinary lateral view of the skull, for instance, is useless in such a case, as the penetration required to pass through the comparatively thick vault would completely blacken out the thin nasal bones. A much less degree of

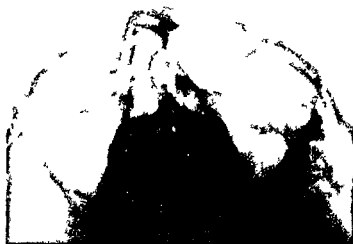


FIG 315 Same case as Fig 314 This view shows lateral displacement of the nasal bones

penetration and a much shorter exposure must therefore be used and the resulting radiogram should clearly show the outlines of the soft tissues in addition to the nasal bones as then it is possible to see the deformity of contour of the soft tissues in its relation to the underlying bony injury

The axial view is taken with the chin resting on the film and

the head thrown back in the occipito mental position the tube being tilted 30 degrees towards the feet This causes the central ray to pass axially down the nose giving one an end on view It also gives a good view of the lower orbital margin

### THE MALAR BONES

Fractures of the malar bones may or may not accompany fractures of the nasal bones depending on whether the injury is caused by a blow from in front smashing in the bridge of the nose and expending its force on the malar bones or whether the blow is an oblique one directly on to the malar bone

Next to fractures of the nasal bones this is the commonest type of facial bone injury but fractures actually through the body of the malar bone are comparatively rare for the following reason the bone is roughly star shaped with a strong central body and four comparatively weak processes two to the maxilla one to unite with the frontal bone and one with the zygoma The body is supported entirely by these processes Therefore when a blow of sufficient force is applied to the body of the malar bone the processes bend as far as they are able and then they either break or the synchondroses become widened The frontal process is strong and unites with the malar process of the frontal bone by a synchondrosis supported behind by the outer wall of the orbit As a result therefore there is usually no fracture of this process but simply a widening of the fronto malar synchondrosis which may be seen in the usual occipito mental view taken to show the malar bones Unless the observer appreciates the significance of this widening of the synchondroses it and the accompanying damage to the other processes of the malar bone may

easily be overlooked. It should be borne in mind that no appreciable widening of this synchondrosis can take place without some degree of rotation of the malar bone and consequent fracture of one or more of its other processes. Not infrequently this rotation of the body of the malar bone may be so great as to tilt and raise the floor of the orbit with permanent damage to the eye. The synchondrosis between the malar and maxillary portions of the lower orbital margin practically never widens as a result of injury, as the weakest point here is the infra-orbital foramen and consequently a fracture usually occurs through this point.

Again the maxillary process forming the outer wall of the antrum is thinned out by the development of this sinus and a fracture occurs easily here. In about 30 per cent of such cases the zygomatic process escapes injury, as there is a certain amount of spring in the zygoma to take up the shock.

These secondary fractures not being fundamental to the rays are frequently concealed in the occipito mental view either in regard to their actual presence or in regard to the degree of deformity caused by them and often the only sign one sees of injury in the occipito mental view is this widening of the fronto malar synchondrosis and possibly opacity of the antrum due to effusion of blood into it (Fig 316). In this case one sees the widened fronto malar synchondrosis but for this to have widened there must have been a fracture or fractures of the other processes. One also notices

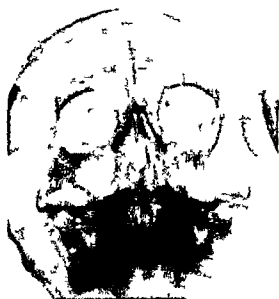


Fig 316 —Fracture of the malar bone (occipito mental view). Note widening of left fronto malar synchondrosis.



Fig 31 —Same case as Fig 316 30 degrees occipito mental view. Note fracture of left lower orbital margin, facial wall of left antrum and comminuted left zygoma.

that the left antrum is opaque and, in addition, there is a fracture of the tubercle of the zygoma, so that the force applied must have been severe. The degree of severity, however, cannot be appreciated in this view, and it is only by directing the rays at right angles to the line of force, as in the 30 degrees occipito mental view, that one sees that the lower orbital margin has been fractured through the foramen and the whole of the malar portion displaced bodily backward into the antrum (Fig 317). In spite of the deformity of the bony parts, however, when this case was X rayed a few days after the injury the swelling of the soft parts completely concealed the depression. Taken early such a deformity can easily be corrected by the

Gillies' operation, but, if not recognised until union has commenced to take place it is extremely difficult and often impossible, to correct it.

### THE ZYGOMA

Fractures of the zygoma may be the result of indirect violence through the malar bone, or may be the result of direct violence, in which case it is frequently comminuted. The best view to show this is the vertico mental view, the rays being centred at right angles to the vertex. Fractures of the zygoma are in a large proportion of cases comminuted (Fig 318).



FIG 318.—Fracture of left zygoma

### THE MANDIBLE

Fractures close to the symphysis are often concealed in an antero posterior view as the shadow of the cervical spine is superimposed. They are best shown by rotating the patient's head slightly to one side and directing the rays obliquely upward on to the film.

Again a fracture of the horizontal ramus is best shown by directing the rays obliquely upward so as to throw the uninjured jaw clear of the injured side.

The temporo mandibular joint is well seen if the head is placed in the lateral position with the injured side against the film and the rays directed downwards at an angle of 30 degrees, centring on the external auditory meatus nearest the film.

## PART THREE

### SECTION V

## INFLAMMATORY DISEASES OF BONES AND JOINTS

### CHAPTER XXVII

#### PERIOSTITIS AND OSTEOMYELITIS

By E DUFF GRAY M.D. F.R. D.M.R.E.

#### PERIOSTITIS

INFLAMMATION of the periosteum is recognisable radiographically only when new bone has been formed and this does not take place until after an interval of at least two or three weeks from the commencement of the inflammatory process.

Ossifying periostitis when it occurs as an isolated affection may be caused by infection from the blood stream by spread of a neighbouring soft tissue inflammation or as the result of trauma with superadded infection.

The X ray appearances are those of a linear opacity curving slightly away from the cortex of the bone and separated from it by a narrow clear space which fills in later resulting in a uniformly dense structureless area of new bone formation continuous with the underlying cortex.

A strongly developed bony ridge at the site of a muscular insertion may give a radiographic appearance very similar to the deposition of periosteal bone and this possibility must be borne in mind particularly when examining films of the metacarpals metatarsals and fibula.

Periostitis plays an important part in osteomyelitis of a long bone and its various appearances in that condition will be considered later. It may be mentioned here that when there is a diffuse osteomyelitis of one of the bones of the forearm or leg the neighbouring parallel bone often shows well marked periostitis (Fig. 327).

The possibility of periosteal bone formation being due to syphilis or typhoid should always be considered and so also should the question of early Ewing's sarcoma. As regards the latter it should be noted that the appearance of successive layers of periosteal new bone (the so called onion peel reaction) is sometimes seen in purely inflammatory lesions.

#### OSTEOMYELITIS

Osteomyelitis as a general term refers to any inflammatory lesion involving cancellous bone but when used without qualification it is generally

taken to mean that form of pyogenic bone inflammation which is secondary to a blood infection and this will be considered under the heading *Acute Hæmatogenous Osteomyelitis*

Less commonly osteomyelitis occurs as an extension from a neighbouring infective process as for example the involvement of phalanges in whitlow and the involvement of the skull in septic processes of the accessory nasal sinuses or the mastoids

Osteomyelitis may also be the result of organisms introduced directly from without as in the case of infected compound fractures and amputation stumps

### **Acute Hæmatogenous Osteomyelitis**

This disease may occur at any age but is most commonly met with in children between the ages of 5 and 15 the sex incidence showing a well marked preponderance of males. The bones most commonly affected are the tibia femur and humerus

**Pathological Features**—The sequence of events in a typical case is as follows. The infecting organism (usually the staphylococcus aureus) gains entrance to the body through the skin throat or alimentary tract and produces a general blood infection (which may be quite transitory and may pass unnoticed)

Organisms in embolic form are arrested in some part of the osseous circulation. This may be in the nutrient artery at its entrance to the bone or in one of its major branches but as a rule the organisms are arrested in the terminal branches in the juxta epiphyseal region and hence the most frequent site of the bone focus is in the cancellous part of the metaphysis of a long bone close to the epiphyseal cartilage

Pus is formed at the original focus in the metaphysis and causes local cancellous necrosis. The infection is rarely arrested at this stage but tends to spread first towards the surface of the bone and later towards the medullary cavity. When pus has penetrated the cortex a subperiosteal abscess is formed which raises the periosteum over wide areas of bone. The elevation of the periosteum interferes with the vascular supply of the cortex and this results in the death of those parts of the compact bone of the shaft which are deprived of their blood supply

Deeper parts of the bone may have their blood supply cut off by thrombosis in branches of the nutrient artery and it is probable that inflammatory exudate under tension in the bony canals produces the same result by compressing the blood vessels. The devascularised areas of bone die and unless they are extremely small have to be removed later as sequestra

Spread of infection from the original focus to the medullary cavity and along the shaft depends on at least three factors (1) direct extension (2) indirect extension via the subperiosteal space and (3) spreading thrombosis

Direct extension undoubtedly plays a considerable part though according to *Starr* a more important route is a backward spread of infection towards the

medulla from the collection of pus under the periosteum the infection passing through the Haversian and Volkmann's canals in the cortex. Amongst other points which he regards as favouring this theory is the characteristic 'spotty' involvement of the cortical areas as seen radiographically which suggests that the organisms pass through the cortex at various places. Hensky stresses the importance of spreading thrombosis in the vascular channels as a cause of extension.

Except in fulminant cases reparative processes become manifest at an early stage even while the destructive processes are still progressing. New bone is formed under the rused periosteum and eventually encloses the diseased part of the shaft more or less completely. This subperiosteal deposit called an involucrum is perforated at many places by openings known as clovæ through which discharges track into the soft tissues.

Another reparative process is concerned with the removal of the gross necrotic areas in the bone. These necrotic areas are gradually separated from the still living parts of the bone by the action of granulation tissue and are known as sequestra. Small sequestra may be extruded through the clovæ but many have to be removed surgically if they are confined by the involucrum.

Later there is a gradual reconstruction of the shaft by compact endosteal bone. Eventually the subperiosteal bone tends to be absorbed the dense endosteal bone becomes more porous and the original architecture of the bone is restored to a large extent.

**Radiological Features**—For at least the first eight to twelve days of the disease there are no radiological abnormalities demonstrable in the affected bone. The first visible change is generally the appearance on the surface of the bone of a regular linear opacity blending with the cortex above and below but elsewhere separated from the shaft by a narrow clear space. Tangential views may be necessary to demonstrate the earliest stage of this periosteal reaction. At about the same time or even possibly earlier a somewhat ill-defined rounded or oval area of rarefaction may be seen in the metaphyseal region.

The first visible changes are seen earlier in children than in adults—sometimes within one week—but an absolutely normal appearance of bone may be seen during the first three weeks even in children and during this period the



FIG. 319. Acute osteomyelitis of humerus. One month after clinical onset of disease. Areas of cortical necrosis in shaft and subperiosteal new bone formation.

disease cannot be excluded by radiological means. Within a short time in addition to the general hyperæmic rarefaction which is soon manifest, irregular areas of increased translucency are visible extending widely from the original



FIG. 320.—Acute osteomyelitis of tibia. Extensive necrosis in shaft. Irregular periosteal bone forming involucrum.

The subperiosteal new bone increases greatly in amount to form the involucrum and its surface becomes very irregular in outline. Sooner or later it is seen that the destructive processes have ceased to spread and from now on during the usually long drawn-out reparative phase the X ray appearances alter much more slowly. With the subsidence

focus in the metaphysis along the shaft and also invading the cortex. These rarefied areas are not very sharply defined, and they show a tendency to coalesce. They are often elongated in the long axis of the bone, especially in the cortex. Between the rarefied areas there are regions of normal bone density which represent areas of bone whose blood supply has been cut off—consequently they do not share in the general hyperæmic osteoporosis and appear dense by contrast. Many of these areas will later be recognised as sequestra. It is sometimes possible to demonstrate radiographically collections of pus in the soft tissues while the destructive process is advancing in the shaft.



FIG. 321.—Late stage of acute osteomyelitis of humerus. Involucrum fully formed. The compactum of the old shaft seen within the involucrum.



of the intense hyperæmia the bone structure as a whole becomes much more normal in appearance regaining its density and sharpness. Cavities become better defined and *sequestra* become more clearly recognisable. The subperiosteal new bone, while diminishing in amount, increases in



FIG. 322—Chronic stage of acute osteomyelitis of femur. Completely separated sequestrum lying in cavity partly formed by involucrum. The lower end of the sequestrum projects through a cloaca.



FIG. 323—Subacute osteomyelitis of tibia. Very little necrosis—well marked periosteal reaction.

density. It becomes more regular in outline and more uniform in structure. At this stage cloacæ are often clearly visible, leading to cavities in the cortex containing sequestra. The shaft is gradually remodelled and replaced by bone that is much denser than normal to start with. The change to cancellous bone is slow and rarely complete.

Except in very young children in whom the infection has been chiefly subperiosteal osteomyelitis generally leaves bone changes which are recognisable radiographically throughout the patient's life. These changes consist of irregularity of bony architecture with areas showing varying degrees of sclerosis, sharply defined cavities and bone defects. Localised recrudescence of active disease may occur even after an interval of several years and a X-ray examination may show the presence of a sequestrum or recent periosteal reaction. Sometimes however a small sequestrum may be entirely obscured by an area of sclerosis and no signs of activity may be demonstrable.

The diagnosis of acute hæmatogenous osteomyelitis is rarely in doubt.



FIG. 324.—Subacute osteomyelitis: cortical abscess. The lateral view shows the abscess to be superficial in the cortex posteriorly, not a central medullary abscess (cf FIG. 316).

clinically and it is important to realise that X-ray examination does not help in early diagnosis. It must be remembered that throughout the acute progressive stage of the disease the X-ray appearances lag behind the corresponding pathological processes. This is not to deny the usefulness of X-ray examination in providing information of great clinical value in the later stages. Radiological evidence is of particular assistance in determining the sites of sequestra and in deciding whether they are sufficiently free for removal.

### Subacute and Chronic Osteomyelitis

While most cases of hæmatogenous osteomyelitis have an acute onset with high pyrexia and intense pain and tenderness in the affected bone, cases are not infrequently met with in which the onset is less abrupt and in which when

first examined radiographically quite definite bone changes are manifest. The X ray appearances may be similar to those of a fully developed case of acute osteomyelitis with widespread bone destruction and periosteal reaction but frequently the bone destruction is less extensive and may even be restricted to a relatively small area in the cortex. An example of such a subacute lesion is illustrated in Fig. 323. The periosteal reaction in the more localised cases tends to consist of the laying down often in layers of denser and more regular bone. Further the initial lesion is quite frequently in the shaft of the bone as opposed to the almost invariable metaphyseal site in acute osteomyelitis. Such cases of low grade virulence which run a chronic course are more common in young adults and older children than in the young.

In some of these chronic cases great difficulty may be experienced in the differential diagnosis between an inflammatory lesion and neoplasm and indeed the distinction is often quite impossible radiologically from a single examination.

Another form of lesion sometimes met with is shown in Fig. 324. The condition here is a subacute bone abscess in the cortex near the upper end of the tibia. The primary site of the arrest of organisms in this case must have been in a periosteal vessel supplying that part of the cortex.

An interesting example of chronic osteomyelitis due to a neighbouring source of infection is illustrated in Fig. 325. In this case there was a large tropical ulcer overlying the anterior surface of the tibia.

**Brodie's Abscess**—This is a rather rare form of chronic osteomyelitis in which a chronic abscess develops in the cancellous bone near the end of a long bone most commonly the tibia (Fig. 326). It generally occurs in young males between the ages of 14 and 25. There may be a history of a previous osteomyelitis though not necessarily in the same bone. The abscess appears as a translucent area often oval with its long axis in the axis of the bone. It may show sclerosis of its margin and possibly a small sequestrum. There is as a rule no periosteal reaction but the cortical bone is generally increased in extent and density. The lesion may lie some distance from



FIG. 325. Chronic osteomyelitis of tibia. It results from spread of infection from a tropical ulcer situated immediately over the periosteal node.



FIG. 326.—Brodie's abscess, upper end of tibia. The cavity was centrally placed in the cancellous tissue and surrounded by a zone of sclerosis.



FIG. 327.—Osteomyelitis of radius in an adult. The infection has spread longitudinally in the bone and involved the articular surfaces. (Note periosteal reaction on radial surface of ulna.)

the epiphysis, owing to growth in length of the bone since the abscess originated.

The differential diagnosis from benign bone cyst is generally easy, as in the latter condition the lesion is usually larger, there is more expansion, and the cortex is thinned.

*Brailsford* has recently published a valuable study of sixty-two cases of chronic bone abscess. In his series, the most common site of the lesion was near the lower end of the tibia. In this situation, the shape of the abscess, especially in the more chronic types, was frequently found to be elongated rather than rounded or oval. In many cases the length of the cavity was one inch or more as against a width of about one quarter of an inch.

**Sclerosing Osteomyelitis (Garre)**—This is a rare form of chronic non suppurative osteomyelitis generally affecting either the tibia or femur in older children and young adults. It is characterised by a diffuse area of sclerosis near the end of the diaphysis, gradually fading off into normal bone.

The cortex is increased in extent and density, and the medullary cavity



FIG. 328.—Osteomyelitis of frontal bone in an adult. The infection spread serpigiously in the diaphysis and produced in three weeks the worm-eaten appearance seen in the figure. Serial films showed progressive widening of the channels of infection. Later a few small sequestra were exfoliated from the outer table. Recovery.

may be so much encroached upon as to be unrecognisable. A somewhat fusiform expansion of the shaft results and there is generally slight roughening of the surface. The X ray appearances in the early stages of some forms of Ewing's tumour may be indistinguishable from this condition.

#### Complications and Sequelæ of Osteomyelitis

**Involvement of the Epiphysis**—In the typical case of osteomyelitis in a child the infection, very rarely spreads from the metaphysis to the epiphysis

and this is accounted for by three anatomical features. Firstly, the epiphyseal cartilage is a strong barrier against direct extension. Secondly, the subperiosteal collection of pus does not tend to spread towards the epiphysis because the deep layer of the periosteum is firmly adherent to the circumference of the cartilage. Thirdly, the blood supply to the epiphysis is practically separated from that of the diaphysis.

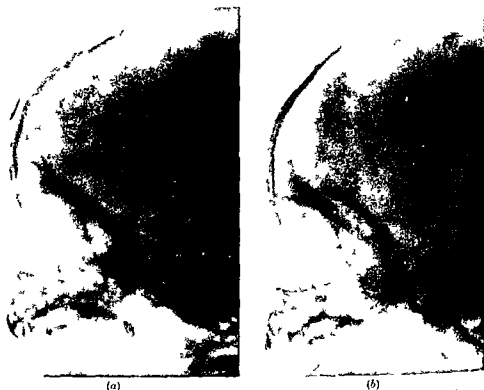


FIG. 329 Osteomyelitis of frontal bone in an infant. (a) Six weeks after infection, shown subperiosteal new bone formation and erosion of the underlying vault. (b) Stage of recovery five months later.

In adults these reasons do not apply and there is no bar to the involvement of the articular end of a long bone (Fig. 327).

Primary involvement of the epiphysis is rare but not unknown. It sometimes occurs in the upper femoral epiphysis in young children and invariably leads to a pyogenic arthritis of the hip joint.

**Involvement of Neighbouring Joints**—This is not very common but may be expected in those joints where the capsule extends over the adjacent metaphysis. The upper and lower metaphyses of the femur are both partially intracapsular. Other examples are the lower metaphyses of the tibia, radius and humerus and the upper metaphysis of the ulna. *Pyrah* and *Pain* comment on the tendency to infection of the knee joint in cases of osteomyelitis.

in the upper end of the tibia. They consider that this is frequently due to inadequate drainage of the upper limit of the subperiosteal collection of pus and emphasise the importance of carrying the incision in the periosteum right up to the epiphyseal line. The radiological signs of joint involvement are considered under the section on Acute Arthritis. Involvement of the epiphysis will naturally lead to joint infection, but, as already stated, this is uncommon.

**Alteration of Growth of Bone.**—Very occasionally the epiphyseal cartilage is damaged by the infection and subsequent growth is retarded or arrested. A more common result from osteomyelitis is overgrowth of the affected bone, an effect which is presumed to be due to hyperæmia. An increase in length of 1 inch is not at all unusual when there has been widespread diaphyseal involvement.

### Special Sites of Osteomyelitis

**Skull.**—Hæmatogenous osteomyelitis is extremely rare. The infection is usually introduced from without or is an extension from local suppuration. The bones of the skull, except in infants, show practically no reparative power, and there is never any periosteal reaction as seen in the long bones.

(1) **LOCALISED FORM**, as a result of an infected scalp wound or a suppurating hæmatoma. This shows radiographically as a limited area of superficial necrosis with sequestration of a thin layer of the outer table.

(2) **SPREADING FORM**.—This generally affects the frontal bone, and originates from a frontal sinus infection (Figs. 328 and 329). The extension of the disease takes place in the diploë and is occasioned by spreading thrombosis in the diploic veins. Not until a late stage is the inner table involved. The outer table has a worm-eaten appearance, and small sequestra may be seen. Subacute and chronic forms of osteomyelitis sometimes occur after typhoid. Sequestration is rare and there is often a good deal of sclerosis.

**Lower Jaw.**—Hæmatogenous osteomyelitis is rare in this bone, though it does sometimes originate in the neighbourhood of an unerupted tooth.

As a rule, osteomyelitis is the result of a spread of infection from a fracture or from an infected tooth socket. Periosteal reaction is well marked along the inferior surface and in the neighbourhood of the angle. Sequestra tend to be either relatively small or grossly large. The former type is seen along the alveolar margin, and the latter occurs at the angle and in the inferior part of the symphysis. The lower jaw has remarkable reparative power, and there is frequently very little residual deformity after even extensive necrosis.

**Phalanges.**—Bony involvement, especially of the distal phalanx, is a common complication of septic finger. There is sometimes intense hyperæmic rarefaction: so much so, that the shadow of the phalanx may disappear entirely, and it is difficult to tell the amount of bone destruction. In many cases, once adequate drainage has been established, a reappearance of bone structure is visible.

In other cases the inflammatory process in the pulp of the finger spreads directly to the phalanx and causes bone destruction, which rapidly spreads through the whole width of the phalanx. The extreme tip of the phalanx has some independent blood-supply and tends to survive for a time, as does also the base of the phalanx with the articular surface, which gets some blood-supply from the joint capsule. The interphalangeal joint is very apt to be involved eventually. There is no periosteal reaction, as a rule, for the periosteum has been destroyed by the neighbouring inflammatory process in the soft tissues. Multiple small sequestra are sometimes seen; more often there is complete absorption of the affected part of the phalanx.

**Spine.**—Acute hæmatogenous osteomyelitis is generally of such a fulminant and fatal character that X-ray examination is rarely of any diagnostic assistance.



FIG 330.—Acute osteomyelitis of the os calcis. Early stage.

The less acute and chronic forms are very apt to be mistaken for tuberculous disease, and the distinction is often not possible in the early stages, when the radiological signs are merely diminution of an intervertebral space, with some irregularity of the adjacent surfaces of two vertebræ. The following points may be of assistance in favouring the diagnosis of staphylococcal osteomyelitis:

- (1) There is less rarefaction of the affected vertebral bodies than in tuberculous disease. Indeed, sometimes there is an appearance of sclerosis that may even suggest malignant involvement.
- (2) There is less vertebral collapse.
- (3) Bony bridges are thrown out from the edges of the bodies.
- (4) Bony fusion between the adjacent vertebræ tends to occur after a relatively short period of immobilisation.

**Pelvis.**—Subacute and chronic cases are not infrequently met with. The commonest site is the upper part of the ilium near the epiphysis for the crest. The disease spreads in the blade of the ilium and may involve the sacro-iliac joint. There is a great tendency for the suppuration to extend widely in the



soft tissues Radiographically, there is generally visible a large area of irregular bone destruction with multiple sequestra Areas of bone sclerosis are often seen near the periphery of the lesion, but there is relatively little evidence of bony repair, and any large bone defect remains permanently

Many of these cases are clinically obscure and the first evidence of a lesion may be the appearance of a discharging skin sinus in the buttock or region of the hip In addition to ordinary radiography of the pelvis it may be necessary to outline the sinus with an opaque medium

**Tarsus**—This is not a very rare site for hæmatogenous osteomyelitis The os calcis is more often involved primarily than the other bones (Fig 330) Extensive necrosis and multiple small sequestra are demonstrable radiographically, without evidence of periosteal reaction There is a great likelihood of spread of the infection to the neighbouring tarsal bones with involvement of the joints

**Amputation Stumps**—Infection of an amputation stump may produce inflammatory changes in the cut end of the bone, but the process does not usually spread far, and there is relatively little periosteal reaction A sequestrum, if formed, is either conical or of ring shape

**Infected Compound Fractures**—After an interval of two or three weeks typical areas of bone destruction are seen in the fragments The infection may spread throughout the shaft but as a rule, does not extend more than a few inches from the site of fracture in either direction Conical or ring sequestra may form as in amputation stumps If the fracture is comminuted and if the periosteum attached to the separate fragments is involved in the infected area these fragments die and remain dense by contrast with the surrounding osteoporosis Periosteal callus is formed, except where the periosteum has been destroyed Occasionally no periosteal callus is developed and a pseudarthrosis is the final result, but, as a rule, the failure of callus formation is confined to one aspect of the fracture, corresponding to the region of infection

#### REFERENCES

- BRAILS福德 J F, 'Brodie's Abscess and its Differential Diagnosis,' *Brit Med Journ*, 1938 II, 119  
PYRAH, L N, and PAEN, A B, 'Acute Infective Osteomyelitis,' *Brit Journ Surg*, 1933, XX, 590  
STARR, C L, 'Acute Hæmatogenous Osteomyelitis,' *Arch Surg*, 1922, IV, 567  
WILENSKY, A O, 'Osteomyelitis,' New York, 1934.

## CHAPTER XXVIII

### TUBERCULOSIS OF BONE

BY

F CAMBELL GOLDING M.B., M.R.C.P. D.M.R.E.

THE TUBERCLE bacillus tends to be deposited towards the ends of bones in the spongiosa. This may be explained by the embolic theory of the occlusion of end arteries.

Several types of lesion with subgroups have been described by various authors. Tuberculosis of bone is a disease of protean radiological manifestation and a concise classification of types is difficult. The reaction of the cortex and medulla to the infection differs and a classification based on the X-ray appearances will depend on the relative extent of involvement of these parts.

#### PATHOLOGY

**THE FORMATION OF FOLLICLES**—The follicle consists of a collection of epithelioid cells surrounded by lymphocytes. Some giant cells are usually present. The follicles coalesce to form nodules with a poor blood-supply. The endothelium of neighbouring vessels becomes swollen and the centre of the nodule tends to caseate as it enlarges.

**REPLACEMENT OF THE MARROW BY GRANULATION AND FIBROUS TISSUE**—Some granulation tissue forms between tuberculous nodules. The tissue may invade a varying proportion of the shaft. In some cases the amount of granulation tissue and erosion is considerable and necrosis is slight.

**ABSORPTION OF BONE AND FORMATION OF SEQUESTRA**—The lamellæ of bone are destroyed and the dead fragments are absorbed, but if a large part of the cancellous tissue is involved a sequestrum forms, invaded by granulation tissue. The necrotic area is surrounded by a fibrous reaction.

**The Radiological Evidence of these Changes**—The presence of the tubercle bacillus with its cellular reaction induces a state of hyperæmia. The cancellous bone undergoes osteoporosis and in certain sites the periosteum may be raised by serous effusion. There is a localized destruction of cancellous bone which is visible as a rarefied area. Small sequestra may be visible but they are irregular and ill-defined compared with the sequestra of septic bone lesions. If the cortex is osteoporotic and the tension within the bone is raised a local expansion tends to occur. The excess of calcium under the periosteum may be used to form new bone. Periostitis is less common than in septic bone lesions.

The lesion is not characterised by a peripheral sclerosis. One exception to this statement is not infrequently encountered in tuberculosis of the os calcis.

Four pathological types of lesion are found. They occur in a decreasing order of frequency, as follows:

- (1) A localised osteomyelitis with caseation
- (2) An extensive osteomyelitis or granulating form
- (3) A tuberculous periostitis
- (4) A cystic form (osteitis tuberculosa multiplex cystoides)

Radiologically, it is convenient for descriptive purposes to divide the disease into tuberculosis of the short, the long, and the irregular and flat bones.

### TUBERCULOSIS OF THE SHORT BONES

**Tuberculous Dactylitis (Spina Ventosa)**—The onset occurs typically in infancy; it is comparatively rare after the age of 10 years. Several short bones are frequently involved, and the condition may be associated with other tuberculous lesions of bone.

The earliest evidence of involvement is shown by periosteal elevation with new bone formation (Fig 331). This can often be detected before changes are visible within the cortex. Later, the cancellous bone becomes rarefied and partly destroyed. Within the area of necrosis small dense particles or definite sequestra may be visible. The cortex tends to expand and with the deposit of new bone under the periosteum gives the fusiform shape associated with dactylitis (Fig 332). Perforation of the cortex may occur with resulting sinus formation.



FIG 331—The slight periostitis visible on the proximal phalanx preceded tuberculous destruction of the medulla.

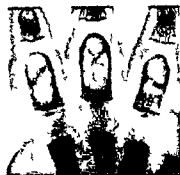


FIG 332—Tuberculous dactylitis with out sequestra or sinus formation.

**DIFFERENTIAL DIAGNOSIS**—See Syphilitic Dactylitis.

**Osteitis Tuberculosa Multiplex Cystoides (Jungling)**—A condition characterised by circumscribed areas of destruction in the short bones frequently associated with skin lesions and involvement of lymphatic tissue.

**CLINICAL FEATURES**—A fusiform swelling over the affected site is often the only local evidence of disease. The condition usually begins in adolescence. Males are more frequently affected than females. The disease may be associated with enlargement of the lymphatic glands or Boeck's sarcoid. The skin

lesions are situated over the affected site or at some distance away and occasionally they are absent

The course of the disease is often symptomless in spite of the extent of the bony lesions. Pain when present is usually an early feature. The progress of the condition is slow with a tendency in some cases to spontaneous recovery. Inoculation of a guinea pig may give negative results but the tuberculous origin of the process is stated to have been proved in many cases (*Jacobsen Ayre Stalman*). The lesions in the guinea pig usually take longer to develop than in other tuberculous diseases. Until further evidence

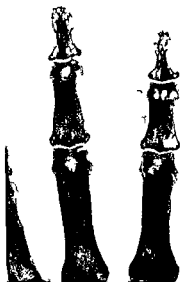


FIG 333 Osteitis tuberculosa multiplex cystica. The central situation of the cystic areas in the end of the bone is a typical feature



FIG 334 —The localized necrotic form of tuberculosis. The condition has spread through the epiphyseal cartilage. A sequestrum is present

is produced it is suggested that the condition be regarded as an infective granuloma of unknown origin

**RADIOGRAPHIC APPEARANCES**—The cysts in the hands and feet vary considerably in size. They appear as multiple transradiant areas in the shaft of the bone. The smaller cysts are usually rounded and clearly defined (Fig 333). In some cases the cysts fuse to form large oval areas and the joint surfaces may be involved. An appearance is then produced resembling the punched out areas of gout.

Within the last few years some examples of the disease have been reported in which these lesions were found in the long bones (*van Alstyne and Gowen Sanes and Smith Jacobsen Connolly*). In these patients the cystic

areas are stated to be much larger than in the short bones. These cysts may be confused with the localised form of tuberculosis of long bones. Several of these cases are open to this criticism.

### TUBERCULOSIS OF LONG BONES

**Localised Form**—One type of lesion begins in infancy as an area of rarefaction and destruction in the region of the metaphysis. This enlarges



FIG. 333.—A peripheral tuberculous abscess containing a sequestrum.



FIG. 334.—A large central tuberculous abscess, containing small sequestra with slight periosteal reaction.

by the local destruction of cancellous bone, which may be absorbed, or may form an irregular sequestrum. In this situation it is of surgical importance, as it is capable of penetrating the epiphyseal cartilage and involving the joint. The fact that the process is not obstructed by the epiphyseal cartilage is a useful point in diagnosis (Fig. 334).



FIG 337 The extensive destruction of the centrum with dense areas of necrosis. The lesion occasionally extends into the hip joint

An abscess forms which is usually central but may be peripheral. The lesion may also occur in the diaphysis. In this group as in other tuberculous lesions of bone it can be seen that with the involvement of cancellous bone necrosis is the predominant feature (Figs 335 and 336) but when cortical bone is affected new

bone tends to form under the periosteum. The latter is more obvious in localised lesions of the diaphysis which are also more often associated with expansion of the cortex than lesions near the metaphysis.

**Infiltrating Form**—In a second type of the disease extensive infiltration of the shaft by granulation tissue is the predominant feature. The appearances are those of an osteomyelitis in which destruction of cancellous tissue is more obvious than is sclerosis.

In some cases of cortical involvement an involucrum forms but in contrast to that of pyogenic osteomyelitis it is frequently rarefied or laminated with very small areas of destruction within it.

### TUBERCULOSIS OF THE FLAT AND IRREGULAR BONES

The lesions are similar in type to those seen in the juxtaepiphyseal region of long bones that is a localised necrosis is the typical feature (Fig 337).

The lesions in the skull are frequently multiple. The frontal and parietal bones are most often affected. The infection appears to



FIG 338—There is a localised kyphosis with diminution of the disc space and marginal irregularity. The destruction extends deeply into the lower vertebra. The central sequestrum

arise in the diploc with circular erosion of both tables. Sequestrum formation is common.

Tuberculosis of the irregular bones is of some importance as it includes Pott's disease. Changes of a similar type may be seen in the tarsal bones especially the astragalus and os calcis. Tuberculosis of the greater trochanter also bears more resemblance to this type than to the other lesions of long bones.

Tuberculosis of the Spine is the commonest of all tuberculous lesions of bone. The onset is usually in infancy or adolescence. The maximum incidence is before 5 years. The aged however are not exempt.

In a series of 270 patients seen at the Royal National Orthopaedic Hospital the sex incidence was equal until 15 years. From 1 to 15 years there were twenty six males and eighty seven females.

Four types can be recognised: (1) metaphyseal or marginal; (2) central; (3) subperiosteal; and (4) appendicular.

In Groups 1-3 similar characteristics are manifest. The first evidence



Fig. 339.—The tuberculous process resulted in collapse of two bodies and invasion of a third. A large abscess formed and tracked upwards. Note the aneurysmal phenomenon in the lower dorsal vertebrae.

of the disease is an area of necrosis. The commonest site is adjacent to the disc where it appears as a circumscribed punched out area or as a diffuse erosion of the surface. Narrowing of the disc space is an early and important feature.

The central lesion forms a rounded zone of rarefaction which rapidly extends to the periphery frequently towards the lower disc (Fig. 338). An apparent marginal erosion may in some cases be traced to a central necrosis if a film is obtained which gives good detail.

In addition to these types a spreading periosteal form is sometimes encountered



Fig. 340.—Tuberculous erosion of two vertebrae in an infant.

This is usually anterior but may be on the posterior surface of the bodies. This type may be difficult to detect as bony destruction is minimal. Several bodies are usually involved with abundant suppuration. The disease spreads by tracking under the longitudinal ligament.

In any of these forms a tracking abscess may cause a secondary lesion with collapse of a body some distance away from the original infection. There may also be two primary bone lesions in the spine. The transmitted pulsation of the aorta through an abscess lying on the surface of the bodies may produce the

same mechanical effect as aneurysmal pressure. These changes are confined to the region between Thoracic IV & V (*Ghormley and Bradley*) (Fig 339).

Collapse of a body affects the anterior more than the posterior border and produces a kyphosis or kyphoscoliosis. In some cases the bodies collapse in concertina fashion. Kyphosis is also caused by erosion of vertebral surfaces. Half or more of a body may be lost by this process (Figs 340 and 341).

When there is complete destruction of discs bodies may fuse and it may be difficult to determine the exact number of vertebrae which are involved. This may usually be done by counting the ribs transverse processes or pedicles.

FIG 341 A later stage of the same type as Fig 340 with complete destruction of the lower and wedging of the upper body. A type of lesion more common in infants than in adults.

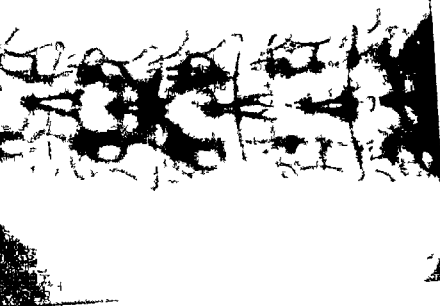
Dislocation is sometimes produced. It is more common in those patients who have marked changes in several vertebrae.

The presence of an abscess may be inferred before calcification takes place within it. In the lumbar region the shadow of the psoas deviates laterally (Fig 342). A paravertebral opacity is visible in the chest by reason of the contrast of the lungs and in the cervical region displacement of soft tissues especially the trachea can be detected. In the later stages large or small areas of calcification are clearly visible in the track or mass of the abscess (Fig 343).

Sclerosis of bone and new bone formation are rare in a tuberculous lesion. Many cases have been published showing these features in the spine. It is not denied that they may occur but in their presence the radiological diagnosis of a tuberculous origin becomes doubtful unless a secondary infection has been added. Sclerosis may be simulated by the calcification of tuberculous debris.







Pl 21a - Total loss of the low part between Pl 1 and Pl 111. The whole is in a series of small areas on the left to right with the spreading of the trees. The w



Pl 21b - Total loss of several verticals along the left side of the image. The w

## REFERENCES

- ALLISON, N *Arch Surg*, 1921, II, 593  
 CHEVALLIER P, and ORNSTEIN E, *Bull Soc Franç derm syph*, 1936, XLIII, 1815  
 CHISHOLM D R, *Canad Med Ass J*, 1935 XXXII, 302  
 GHORVLEY, R K, and BRADLEY, J I, *J Bone Jt Surg*, 1928, X, 796  
 GOFCKERMAN W H *Arch Derm Syph*, 1928 XVIII, 237  
 HECHT P Z *Tuberk*, 1928, LI, 125  
 HERSKOVITS E, *Röntgenpraxis*, 1937, IX, 45  
 JACOBSEN, A W, *J Pediat*, 1936, VIII, 292  
 JUNGLING, O, *Fortschr a d Geb d Ront*, 1920 XXVII, 375, *Beitr klin Chir*, 1928, CXLIII 401  
 KOCH F *Derm Wschr*, 1935, CI 919  
 KREMER, W and WIESSE, O *Die tuberculose der Knochen und Gelenk*, Berlin 1930  
 KYRLE J *Arch f Dermat u Syph*, 1921, XXXIII, 131  
 MAGNUSSON, R *Acta Orthop Scand*, 1935, VI, 93  
 MAYR J K, *Arch f Dermat u Syph*, 1928, CLV, 248  
 SANES S and SMITH, W S, *J Amer Med Ass*, 1934, CII, 1206  
 SCHURER WALDHEIM, F, *Arch klin Chir*, 1930, CXL, 680  
 SORREL, D, *Tuberculose Osseuse et Osteo articulaire*, Paris 1932  
 STALMANN, A *Arch f Klin Chir* 1934, CLXXVIII, 669  
 VAN ALSTYNE G S, and GOWEN, G H *J Bone Jt Surg* 1933 VI, 193  
 VON FRIEDLANDER F, *Dtsch Z Chir*, 1904 LXXIII, 249

## CHAPTER XXV

### TUBERCULOSIS OF JOINTS

BY

F CAMPBELL GOLDING MB MRCP DMRD\*

**TUBERCULOUS DISEASE** is one of the most frequent lesions of joints encountered in routine orthopaedic practice

It is important to bear in mind that the radiological evidence of infection tends to lag behind clinical signs. In the lung it is possible to make an early diagnosis of tuberculosis by reason of the contrast between air filled spaces and tuberculous tissue whereas some time must elapse before the dense cortex and firm network of bone show signs of disease

The radiogram may not give conclusive evidence of the nature of the infective organism but during the stages of invasion progress and repair sufficient evidence may be found on the film to confirm a tentative diagnosis. Many joint infections tend to lose their characteristic features in the years after the process has subsided. In tuberculosis this tendency is intensified in weight bearing joints and in those invaded by secondary organisms

**Age Incidence** —The disease is commonest in infancy reaching its maximum incidence between 3 and 7 years and declining progressively to 30 years after which it is uncommon

**Sex Incidence** —In a series of 840 patients at the Royal National Orthopaedic Hospital who had tuberculosis of the bones and joints the sex incidence was equal apart from tuberculosis of the sacro iliac joint (fourteen females two males) and the older patients with tuberculosis of the hip and knee joints (in whom the ratio was four females to one male)

**Joint Incidence** —In the above series the joints were affected in the order —hip knee tarsus ankle sacro iliac joint elbow shoulder and wrist

**Mode of Infection** —A joint may be involved as a secondary process by the tubercle bacillus in several ways by (a) direct synovial implantation (b) rupture of an osseous focus in the end of the bone (c) extension of a metaphyseal lesion or (d) extension from a lesion in soft tissue (e.g. abscess bursitis or tenosynovitis)

### PATHOLOGY

Involvement of the synovial membrane by tubercles induces hyperaemia and swelling. Granulation tissue forms and spreads. The cartilage is attacked

from above and below it may be perforated or separated as fragments or entirely detached. The calcium content of the bones is lowered by relative hyperæmia and later by disuse. Some endarteritis is present but in contradistinction to syphilitic and certain septic processes it is of secondary importance. The major changes are due to the granulomatous nature of the infection.

The subchondral bone is invaded by granulation tissue and partly destroyed. The amount of destruction varies—it may appear as a solution of contiguous surfaces or as a marginal erosion whilst in other cases the destruction is more extensive and spreads deeply into subchondral bone. In the presence of deep erosions sequestration of bone is liable to occur. There is a tendency for the lesion on one side of the joint to be duplicated on the opposite articular surface. Destruction of the end of the bone with the presence of fluid and laxity of ligaments may cause subluxation of the joint.

As the progress of the disease is checked some healing by fibrous and bony ankylosis occurs. The natural healing of a tuberculous joint especially by bone formation is slow and imperfect. The resorptive process in the cancellous structure induced in the early stages and carried on during the progress of the disease tends to leave a permanent mark in the form of a coarse cancellous network for some distance round the joint.

### RADIOLOGICAL APPEARANCES

The interpretation of the film is based upon the pathology. The two common modes of onset are osteal and synovial. The relative frequency of these at various ages in different joints is disputed.

**Stage of Invasion**—The earliest evidence of invasion is a localised porosis of the bones of the joints. This is not a sign produced by the tubercle bacillus alone but in the event of inconclusive clinical evidence it may be of the utmost importance indicating that the patient should be kept under observation. At this stage the osteoporosis is too slight to allow of successful reproduction in a text book illustration which has therefore not been attempted. To detect this change it is essential to radiograph the opposite joint as a control at the same time and if possible on the same film so as to eliminate any difference of technique. As the outline of the cortex vanishes in bones which are already rarefied a false appearance of widening of the joint space may be produced.

In the event of the primary focus arising in articular bone the course of the disease is the same but the original focus may be visible on the radiogram as a localised area of destruction e.g. in the acetabulum. In many cases it is difficult to determine if the origin has been osseous or synovial.

**Stage of Progress**—The local products of the tubercle bacillus have not the power of dissolving cartilage possessed by those of the organisms associated with the acute septic joint. Destruction of cartilage is brought about by

disturbance of its nutrition. Early ablation of the joint space is not, therefore, a feature of tuberculosis. As the disease progresses, however, some narrowing of the joint space occurs with further changes in subchondral bone. The margins, which were ill defined as a result of superficial erosions, may become deeply pitted with excavated areas as seen in the knee or shoulder joint. Cone or thumb shaped lesions are usually tuberculous.

The amount of fluid formed in the joint varies. In the shoulder and hip joints a dry form called *caries sicca*, sometimes occurs. A large collection of pus may rupture through the skin or into the tissues causing a tracking abscess, the path of the latter may be traced by a deposit of calcium in the inspissated material left by the abscess.

The tension of fluid, together with the laxity of ligaments and eroded margins of the joint, tends to cause subluxation. The direction of the subluxation is determined by muscle spasm. In the knee and elbow this is most frequently backwards, in the wrist forwards, and in the hip upwards. Dislocation when it occurs is usually an early event.

As granulation tissue invades subchondral bone, it separates fluffy fragments, which are usually absorbed. Sequestration of bone is not very common, and is usually evidence of deep erosion. Its recognition is dependent on the difference of density of the two parts. The tuberculous sequestrum frequently retains an attachment to the bone, and does not separate in the same clear way that an osteomyelitic sequestrum separates. Opposing areas of destruction with 'kissing' sequestra are seen typically in the knee and hip.

The absence of sclerosis of articular and subarticular bone and the failure to form new bone are important diagnostic points. Invasion by secondary organisms for example through a sinus, alters the appearance, sclerosis and new bone formation are then added to the destruction.

**Stage of Repair**—Healing is manifested in ankylosis, which is usually fibrous sometimes bony. The continuity of bony trabeculation across the joint is evidence of cure. A false appearance of ankylosis is frequently produced by overlapping bones. Bony ankylosis is seldom complete and there may be localised areas of rarefaction in some part of the joint whose potential activity is suspected. These are often walled off and remain inactive indefinitely. A natural attempt at cure simulating a surgical excision, may be seen in the hip joint. In these cases the diseased head and part of the neck are absorbed leaving a pointed process of bone, the *bec d'oiseau*. A similar process in the acetabulum leads to great enlargement of the cavity, the "ballooned acetabulum".

The radiological evidence of cure is based on

- (1) Return of calcium content in the bones around the joint
- (2) Some definition of the cortex
- (3) Absence of local rarefied areas, or no alteration in the appearance of doubtful rarefactions in a series of films taken over two years

(4) Bony ankylosis which is usually incomplete in the absence of surgical intervention or secondary infection

After healing has taken place some evidence of the tuberculous origin of the process may be retained in the joint. This may be in the form of small irregular structureless deposits of calcium in the region of the joint or a wide cancellous trabeculation in the affected bones or the bones of the joint below the original lesion (for example in the knee in a case of hip joint disease). Some secondary effects of infection are seen in children. These signs which are not confined to the tuberculous joint are as follows

(1) Accelerated growth with actual lengthening of the limb. Valgus and more rarely varus deformity may occur

(2) Increased size of the epiphysis e.g. the capital epiphysis of the femur

(3) Early development of ossific nuclei e.g. in the carpus

(4) Diminution in the width of the cortex and enlargement of the medulla

These features are best demonstrated in the knee joint since growth of the limb is largely dependent on the epiphyses in this region. The continuous hyperæmia of infection facilitates these changes. It may for example also cause enlargement of the patellar nucleus

### TUBERCULOSIS OF INDIVIDUAL JOINTS

Apart from the general features which characterise a tuberculous joint in any situation there are additional points in individual joints which merit description. These are frequently an exaggeration of some stage of the infective process or variations due to the type of joint involved

In the following description no attempt is made to describe the progressive change in the X-ray appearances during the course of the disease in the various joints

**Tuberculosis of the Wrist**—*In infants* the wrist may be infected from the blood stream or by extension from a spina ventosa affecting the second to fifth metacarpals. Extension from a tenosynovitis the first metacarpal or the radius and ulna is not common

The disease process is frequently localised (*Sorrel Sorrel Dejerine*). The cartilaginous covering of the bones which is comparatively thick up to the age of 10 years serves to limit the process and confine the infection to a few bones. Early appearance of the ossific nuclei is often a prominent feature in the affected wrist

*In adults* the infection is more generalised than in infancy. Intense rarefaction of bone with loss of bony detail and contour are the predominant features. Sinuses are common (Figs 344 and 345)

**DIFFERENTIAL DIAGNOSIS**—The rheumatic diseases frequently involve the wrist

Some rarefaction of bones is typical in *rheumatoid arthritis* but localised osteoporosis is not so intense as in tuberculosis. In addition rheumatoid

arthritis tends to involve many joints. If the wrist is affected, the other small joints of the hand are usually involved. The typical features of this disease are multiple joint involvement, some generalised porosis of bone, marginal erosions, and multiple sites of loss of cartilage. Return of bone calcium takes place as the condition becomes quiescent, with bony ankylosis of the carpal joints without sinuses, sequestra, or irregular deposits of calcium in the joint space.

**Tuberculosis of the Elbow.**—*In young children* the disease frequently commences in the extremity of the humerus or ulna, more often the latter. Some bone is destroyed, and a sequestrum is not uncommon in the necrosed area. Periostitis is frequently present. The joint is involved by direct spread of the process.

*In the adult* the radiological changes are usually most marked in the ulna. A good lateral film may show a large area of destruction extending round and under its articular surface. Progressive destruction of the articular surface causes a ballooned appearance of the sigmoid notch, with sharpening of the coronoid and olecranon processes. The radiological changes in the head of

the radius are seldom marked: occasionally it appears to have escaped infection.

Periostitis of the neighbouring bones is not unknown in the adult disease. It is important to recognise that this may occur, although it is rare in a tuberculous joint in any situation. The joint is rarely involved by spread from a tuberculous olecranon bursa (Figs. 346 and 347).

**Tuberculosis of the Shoulder Joint.**—Two forms of the disease may be recognised. One type is associated with superficial erosion of the head, which gives



FIG. 344.—The early stage of tuberculosis of the wrist. Note the ill defined cancellous structure of the carpals, the loss of definition of the cortex, particularly of the os magnum, and the periostitis of some of the metacarpals.



FIG. 345.—A later stage of the disease in an adult.



FIG 346—Active tuberculosis of the elbow affecting the three bones with deep erosion of the sigmoid notch and some periostitis



FIG 347—A later stage of the disease. Note the granulomatous invasion of the ulna.

an appearance of atrophy. In subsequent years the cancellous tissue of the head also becomes more widely spaced and the ultimate appearance suggests a shrivelled head.

In the second form there is considerable destruction of bone, and the even contour of the head is broken by punched out or thumb shaped areas of destruction.

The major changes are always present in the head of the humerus but evidence of erosion of the glenoid fossa is frequently visible although it may be difficult to detect in films of poor quality. Either form of joint disease may be present without the presence of pus but it appears that



FIG 348—Tuberculosis of the shoulder joint in a child with superficial erosion of the glenoid



FIG 349—Caries sicca in an adult with a thumb shaped area of erosion



the relative incidence of caries aces has been exaggerated by frequent description. It is more common in the child than the adult (Figs. 348 and 349).

**DIFFERENTIAL DIAGNOSIS**—The conditions which simulate tuberculosis of the shoulder joint are all rare.



FIG. 349. Advanced tuberculosis of the shoulder joint, as related with a case of tuberculous tuberculosis.

**Rheumatoid arthritis**—Occasionally the moderately advanced stage of rheumatoid arthritis in the shoulder may resemble the late result of a tuberculous joint. In this case the patient is usually an adult. There is an absence of areas of erosion, the irregularity of the articular surface is generalised, and other joints are almost certainly involved.

**Spondylitis ankylopoetica**—Although symptoms are frequently referred to the shoulder joint, radiological evidence of arthritic involvement is very rare. In a series of over 200 patients with this disease only one patient had organic disease of the joint which could be demonstrated. The condition was



FIG 351—The typical changes of early tuberculous of the ankle joint. The joint surfaces appear fluffy and there is porosis of neighbouring bone.

the margins of the joint surface on the radiogram followed by subchondral erosions. The amount of destruction varies if it proceeds unchecked oval lakes form in the region of the joint space and these may contain sequestra or calcified debris. An abscess frequently forms over the site or calcified areas in an abscess may be visible below the joint. Subluxation may occur (Fig 350).

bilateral and consisted of areas of extensive destruction resembling the punched out areas in tuberculosis. The excavations were however larger and the disorganisation of the joint was extensive.

*Osteochondritis*—The changes are confined to the epiphysis and subepiphyseal bone. The epiphysis is fragmented with irregular density and absence of rarefaction of neighbouring bones.

*Septic Arthritis and Osteoarthritis*—These are described elsewhere in this volume.

#### Tuberculosis of the Sacro iliac Joint—

This is a disease of early adult life. Females are more frequently affected than males. The earliest evidence of involvement is loss of the white line which marks



FIG 352—Tuberculosis of the calcaneo-cuboid joint. Some absorption of the joint surfaces has taken place.

**DIFFERENTIAL DIAGNOSIS**—Infection of the joint by other organisms is uncommon. One condition which has to be differentiated is the sacro iliac disease which is associated with *spondylitis ankylopoietica*. In this disease the joint changes are bilateral and some subchondral destruction takes place but there is less evidence of erosion and the area is frequently surrounded by a mild sclerosis of bone.

**Tuberculosis of the Tarsal and Ankle Joints**—A tuberculous infection of the tarsus may be restricted to one joint, may involve several or may be generalised. In a series of eighty-two patients with the condition thirty-six presented themselves for treatment before the end of their fifth year.



FIG. 353. A comparatively rare form of onset of tuberculosis with marginal erosion of the left knee joint and loss of the joint space. The patient also suffered from tuberculosis of the lungs and died one month after this film was taken from milary tuberculosis.

A tarsal infection rarely follows a spurs ventosa of the metatarsals. In the early stages of the disease the rarefaction of bone may be so intense that it is difficult to determine which of the bones is affected. In addition to the usual features of a tuberculous infection (Fig. 351) the disease process sometimes causes a marked dissolution of joint surfaces. A large amount of bone may be absorbed and as a result there is a wide space between the affected bones (Fig. 352). In the later stages of the disease small calcified flecks are frequently retained in the region of the joint space.

In lesions of the ankle and subastragaloid joints a primary focus in bone may be visible on the film. This focus appears as a comparatively large area of rarefaction resembling to some extent a cyst with a central ill-defined sequestrum. It may be situated towards the centre or articular surface of either tarsal bone or in the juxta epiphyseal region of the tibia.

**Tuberculosis of the Knee Joint**—Disease of this joint is common. In any large series of patients tuberculosis of the knee appears in the second place preceded only by tuberculosis of the hip. In a series of 146 patients with tuberculosis of the knee the age and sex incidence corresponded almost exactly with the findings in tuberculosis of the hip joint.

At the onset the disease may be synovial, subchondral or metaphyseal. Occasionally the process commences as a clearly defined rounded erosion just below the margin of the tibial articular surface (Fig. 353).



FIG. 354.—Child aged 9½ years. Disease began at 5 years. Radiologically not active. Three years after this film was taken the appearance was unchanged.



FIG. 355.—Tuberculosis confined to the inner half of the joint. Onset of disease at 4 years. Duration was ten years at the time of the radiogram. Excision of the joint was performed. It showed that the disease was limited to the inner half.

The course of the condition varies greatly. It varies from a chronic disease with little erosion (Fig. 354) to marked destruction and even bony ankylosis. The latter cases are usually associated with sinuses. Severe deformity may also be present with dislocation.

Most authorities agree that once the joint is affected by rupture of an osseous focus full movement is never restored (*Girdlestone*). Some cases of the synovial form pursue a chronic course with absence of bone destruction and conservation of the joint space. In these patients the only radiological evidence of past infection may be the coarse trabeculation of subarticular bone.

In a few rare instances tuberculosis tends to remain localised to one part of a joint in adolescence or in an adult. The sites in which this type of lesion may be found are usually the inner half of the knee joint (Fig. 355), the posterior



FIG 356—Early tuberculosis of the right hip joint. The rarefaction of bone is the only evidence of disease. The right capital epiphysis appears to be enlarged but the leg is rotated slightly.

part of the ankle joint and the articular surfaces of the humerus and ulna. As judged from the radiogram the infection in these cases appears to be of low virulence and does not cause severe destruction.

**DIFFERENTIAL DIAGNOSIS**—Below are tabulated the principal differentiating features of conditions which may simulate tuberculosis of the knee.

*Acute suppurative arthritis*—Onset rapid, early loss of cartilage space, osteoporosis often intense with loss of bony detail in neighbouring bone, sequestration common.

*Non specific Infective Arthritis*—Slow onset, chronic course, cartilage loss incomplete and unequal on the two sides, local sclerosis of subarticular bone and tendency to form new bone in late stages.

*Syphilis*—Evidence of sclerosis or irregular cancellous network indicating presence of osteitis in subarticular bone or presence of periostitis.

*Charcot's Joint*—Increased density of bone, gross atrophic or hypertrophic changes affecting the articular surfaces, numerous dense loose bodies, irregular in size and shape.

**Tuberculosis of the Hip Joint**—Hip disease is the commonest of all tuberculous manifestations in joints. In a series of 270 patients at the Royal National Orthopaedic Hospital the greatest number presented themselves for treatment between the ages of 3 and 7 years. Between the ages of 1 and 11 years there were 106 males and 60 females, whereas from 16 to 60 years there



FIG 357—Evidence of subchondral erosion of the acetabulum and head of the femur—the disease being still active.



FIG 358—Patient age 11 years. Onset of tuberculosis at 3 years. No radiological evidence of activity. The acetabulum has moved upwards. Note the coarse trabeculation of bone. The patient retained 45 degrees of flexion and 50 degrees of abduction till death of shortening.

sufficient bone to enable them to extend into the joint e.g. ischium.

The destructive tendency of the infection is particularly marked in the hip joint (Fig 358). A large part of the head and neck of the femur is frequently absorbed and a similar process in the acetabulum leads to great enlargement of the cavity (Fig 359) or even perforation into the pelvis (Fig 360). Occasionally the malacia induced by the disease results in an intrapelvic protrusion of the acetabulum.

A feature of the later stages of the disease is the presence of a partly calcified abscess which passes in fascial planes through the muscles of the thigh for a varying distance. Sometimes the shaft of the femur itself shows a central line of ap-

were four times as many females as males.

The initial osteoporosis is readily recognised as the joints are so easily compared on the same film (Figs 356 and 357). Apart from the usual synovial and osseous origins the hip is sometimes involved from foci farther removed from the joint. These foci may be in the femoral neck or trochanteric region or occasionally in the ischium. The only factor which appears to govern joint infection is the relative proximity of the focus. The distant foci seldom erode



FIG 359—A ballooned and wandering acetabulum. The condyles at iliac crest and the head of femur being destroyed. The sequestered fragments were absorbed two years later and the neck became short and pointed.

parent osteoporosis due to the presence of tuberculous pus which gravitates down the medulla

**DIFFERENTIAL DIAGNOSIS**—In addition to the conditions considered under the knee joint the following must be taken into account

*Congenital coxa vara*—A varoid deformity with elevation and beaking of the greater trochanter. A vertical translucent zone is present in the neck or an inverted Y shaped defect enclosing a bony fragment. Aplasia of the shaft is common

*Rachitic coxa vara*—This condition is bilateral with broadened irregular epiphyseal lines and rachitic changes elsewhere

*Adolescent coxa vara* shows broadening of the epiphyseal line a change in the outline of the upper margin of head and neck and evidence of downward and backward movement of the capital epiphysis

*Osteochondritis of the Femoral Epiphysis*—Increase in density irregularity in density fragmentation and compression of the epiphysis and alteration in structure of the femoral neck are features in this condition

*Osteomyelitis* frequently affects the upper part of the femur and by reason of the peculiar relationship into the hip joint. This condition may be distinguished from the metaphyseal focus of tuberculosis by the extensive distribution tendency to sclerosis formation of several dense sequestra (which may be large) deposits of subperiosteal bone and dislocation of the joint

The clinical findings in congenital dislocation and the hysterical hip may simulate tuberculosis

*Primary Bone Tumours and Secondary Deposits*—The radiographic features are discussed elsewhere



FIG 360—Old tuberculosis of the hip without evidence of activity. The head is absorbed and the acetabulum is much enlarged with a perforation into the pelvis

## REFERENCES

- GIRDLESTONE G R *Brit Jo in Surg* 1931 XIX 490  
 SORREL D SORREL DEJERINE MMF *Tuberculose Osseuse et Osteo arthralgie* Masson & Cie Paris 1937

## CHAPTER XX

### SYPHILIS OF BONES AND JOINTS

BY

F CAMPBELL GOLDING MB MRCP DMPE

#### EARLY CONGENITAL SYPHILIS OF BONE

THE MANIFESTATIONS of congenital syphilis may be divided into early and late. The early period includes the signs which appear from birth to three or four years.

The radiological examination is of some importance as serological tests are not always reliable especially in early infancy. A high proportion of syphilitic infants have skeletal disease during the first six months often with obscure symptoms or only mild skin eruptions. The bony changes are usually widespread in infancy. A pathological process confined to one bone is not characteristic of syphilis.

Radiological evidence of syphilis is found in the form of osteochondritis or metaphysitis, periostitis and osteitis or osteomyelitis. Osteochondritis is most important as it is frequently present in a recognisable form when the clinical diagnosis is difficult.

#### Pathological Features

The end of the shaft of a normal growing bone consists of resting cartilage. The deeper layers are arranged in regular columns of cartilage cells. These cartilage cells show a maximum growth on the diaphyseal side and the larger cells exhibit signs of degeneration. There is some calcification of the matrix between these cells. This calcified zone is visible on the film as a white line called the zone of temporary calcification. Beneath this region is the area of primary spongy bone a framework ultimately replaced by the permanent osseous trabeculae. Interspersed in the primary and secondary osseous framework are loops of blood vessels.

In syphilitic osteochondritis the preparation for ossification continues in a normal fashion but the completion of the process is delayed. The provisional or temporary zone of calcification therefore becomes wider than normal. The blood vessels are arranged in vertical and transverse systems. Of these the vertical loops are the larger. In the pathological process these are less likely to be obliterated and some ossification takes place around them pro



ducing irregular tongues of osseous tissue which give the saw toothed appearance on the radiogram

Fibrotic change is characteristic of syphilis. It is preceded by vascular round-cell infiltration. The formation of fibrous and granular tissues causes areas of softening. The Haversian canals are widened. Osteoclastic erosion of trabeculae takes place. Displacement of the epiphysis is therefore liable to occur. Areas of syphilitic inflammatory change are



FIG. 361.—Periostitis of both bones with a localized area of osteomyelitis of the inner and upper border of the tibia. Increased density of metaphyseal bone with a narrow zone of decalcification.



FIG. 362.—Periostitis of both bones. Syphilitic osteochondritis or metaphysitis of the lower ends of both bones. Osteomyelitis of the inner and upper border of the tibia.

found in the diaphysis. The essential feature is the replacement of bone by connective tissue in small or conglomerate areas. This reaction may be local, generalised and periosteal or central, at the same time osteoblasts form under the periosteum or round the Haversian canals. The shaft may become very dense without a clear demarcation between cortex and medulla.

### Radiographic Appearances

**OSTEOCHONDRITIS**—This type of lesion is confined to the region of the metaphysis. It was called osteochondritis by Hegner (1870). The di-

is usually made during the first six months of life. The earliest evidence of metaphyseal involvement is an increase in density and depth of the zone of temporary calcification. It appears during the first few weeks (Fig 361). This produces a white line on the film at the extremity of the shaft. This line may measure 3 mm in depth. Unfortunately it is not a sign which is produced by syphilis alone but may be associated with cessation of growth from any cause. In the more severe lesions there is a comparatively broad zone of osteoporosis beneath the white line. A diagnosis of scurvy should not be made, as the patient is too young to have contracted this disease.

In the more active and advanced lesions the margin of the metaphysis becomes irregular. During the first few months the sclerosed and saw-toothed metaphysis is probably the most valuable sign in the diagnosis of syphilis (Fig 362). Other features which may appear are a second zone of osteoporosis beneath and parallel to the band already described, or a separation of the epiphysis. The latter is really a fracture through the metaphyseal bone and is usually accompanied by slight displacement or impaction.

The radiological changes are shown best at the distal end of the radius and ulna and proximal end of the tibia.

**PERIOSTITIS**—Periostitis and osteitis may occur as separate lesions but both are frequently found with osteochondritis in its later stages.

Periostitis is of two types, a mild form which occurs in the first few

months, and a more severe type which occurs later. The latter causes considerable thickening of the shaft by the deposition of bone in layers under the periosteum. Periostitis is a very common lesion and may serve as a signpost directed towards the ultimate diagnosis.

**OSTEOMYELITIS**—Specific osteomyelitis may affect any bone especially the long bones. The lesions may be circumscribed or extend for some distance in the shaft. They appear as irregular areas of rarefaction, necrosis and sclerosis. In some patients the combination of rarefying and condensing osteitis resembles a suppurative osteomyelitis but sequestration is rarer than in septic processes. The clinical symptoms associated with severe bony disease are also much more prominent in suppurative osteomyelitis than in syphilis.

Over the affected region new bone is laid down under the periosteum and



FIG 363—Syphilitic dactylitis in an infant with partial destruction of the proximal phalanx of the middle finger.

the shaft becomes thicker than normal. A localised bilateral area of destruction, affecting the proximal and medial surfaces of the shafts of the tibiae, is pathognomonic of syphilis

Two types of lesion are found in the skull, a necrosing and a condensing osteitis. In the former, both tables are affected and sequestra may form. In the latter, the condition involves the frontal and parietal bones particularly; the bones become sclerosed and thickened, producing the "hot-cross bun" effect. This condition usually appears about the ninth month. Craniotabes occurs in congenital syphilis, but is probably due to concomitant rickets.

Syphilitic dactylitis is very rare (Fig. 363): it affects the metacarpals and



(a)  
Yaws

(b)  
Leprosy.

(c)  
Tuberculosis

FIG. 364.

metatarsals as frequently as the phalanges. It may be found at varying ages, often before the end of the first year. The bone is increased in breadth by the deposition of subperiosteal new bone; it is also increased in density and often in length. There is usually less evidence of necrosis and sequestration than in tuberculosis. In many patients the differentiation of these conditions is very difficult or impossible on the radiographic appearances alone.

#### Differential Diagnosis of Dactylitis

**TUBERCULOSIS**—In this condition there may be some periosteal reaction in the early stage, with central areas of necrosis; the latter may be rounded or multiple irregular areas, which enlarge and destroy the medulla (Fig 364).

The bone increases in width but this is due more to expansion than to new bone formation. Necrosis often results in sequestration.

**YAWS**—The differentiation from syphilis may be impossible especially after treatment. The areas of destruction may be peripheral or central. Periosteal bone is deposited and the shaft is sclerosed between the rarefied areas. The condition responds to treatment.

**LEPROSY**—The bone increases in width with central areas of destruction. There is no sclerosis. Fracture of phalanges may occur from minor trauma followed by marked periosteal bone at the site.

In each of these conditions except that of leprosy the bone may increase in length. In tuberculosis and leprosy it increases in breadth by subperiosteal bone and expansion in syphilis and yaws by subperiosteal bone alone.

### LATE CONGENITAL SYPHILIS OF BONE (SYPHILIS TARDA)

The lesions of late syphilis bear some resemblance to the acquired form and may become apparent over a wide period. The more active lesions are usually found between the ages of 5 and 15 years.

Interstitial keratitis and skeletal changes are amongst the most common manifestations of late congenital syphilis. The bony lesions of early congenital syphilis may undergo spontaneous cure or respond to treatment but the late lesions usually resist treatment and deformities tend to persist. Both periostitis and osteitis occur.

**Periostitis**—The periosteal reaction may take several forms. In the young patient the periosteum may be thickened by lamellated layers which lie parallel to the shaft (Fig 36a). In another form which usually appears later the shaft is increased



FIG 36a Infant, aged 4 years.  
There are several distinct layers of periosteal bone.

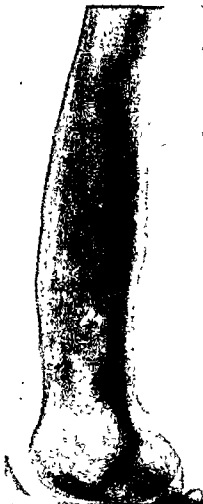


FIG. 366.—Periostitis and increased density of the shaft in a patient aged 27 years, suffering from the congenital form of syphilis.



FIG. 367.—Syphilitic periostitis and osteitis in a patient aged 6 years. Several other bones were affected.

in width by new bone deposited on the convexity of the diaphysis (sabre tibia) (Fig. 366).

**Osteitis or Osteomyelitis.**—In contradistinction to osteochondritis, the body of the shaft is primarily involved, and many patients give a history of previous trauma. Of all the bones, the tibia appears to be most often affected. The changes in the bones may be localised or diffuse. The radiological appearances are those of a process which causes a varying proportion of sclerosis with areas of destruction and sequestration.

The localised lesions are frequently referred to as gummata. They may be periosteal or central.

The diffuse type causes an alteration in cancellous structure the trabeculae become dense and irregular, the cortical bone is thickened and the outline is uneven. Even in the more diffuse lesion, a patchy distribution is evident. This is accentuated by areas of rarefaction. Sequestra are uncommon.

Subperiosteal bone is often deposited and the bone is increased in width (Fig 367). If the process involves a single bone where two are present—for example the tibia and fibula—some bowing may be produced by an increase in length of the affected bone.

### ACQUIRED SYPHILIS OF BONE

The incidence of skeletal changes in the acquired disease has not been investigated as thoroughly as in the congenital form. Periostitis may be found during the secondary period and other bone changes can appear within eighteen months of infection.

The bony manifestations of acquired syphilis are periostitis and osteitis.

Periostitis is the commonest lesion in the acquired disease. Its importance should



FIG 368.—The lace work type of periostitis in the acquired disease. Other bones show similar lesions.

be recognised not because an immediate diagnosis is often possible, but because it should direct the examiner to further investigation, from which a final diagnosis may be reached. Periosteal thickening of the tibia and clavicle are common, but the clinician is liable to gain a false impression of their relative

frequency as they are so easily demonstrated by his examination. The periosteal reaction takes several forms. The commonest type is a simple periosteal thickening; it frequently involves a large part of the shaft and may precede an osteitis. A form which has been described as lace work is more rare; when present it bears diagnostic features (Fig. 368).

Rarely bony spiculation is visible at right angles to the shaft. This feature is generally associated with underlying bony disease of a gummatous type. The spicules of periosteal osteogenic sarcoma are usually longer and better formed; they correspond with the site of a palpable tumour and decrease in size toward the margins of this tumour. In the later stages or in the rapidly advancing growth some of the spicules towards the centre are frequently absorbed giving an appearance of cuffing in the radiogram.

It is advisable to exclude the presence of a varicose ulcer on the shin in any patient exhibiting a localised periostitis.

Osteitis in the acquired form of the disease may be diffuse or localised.

#### IN THE DIFFUSE

FORM the predominant change is sclerosis of bone. The cortex is increased in density and width with patchy or diffuse sclerosis of the shaft. In addition to the sclerosis of cancellous bone the trabeculation is irregular (Figs. 369 and 370). Some rarefied or necrotic areas may be present. Sequestra are small or absent; they are frequently absorbed after formation. The inflammatory change frequently extends to the end of the bone and causes arthritic symptoms.

Bones formed in membrane are particularly

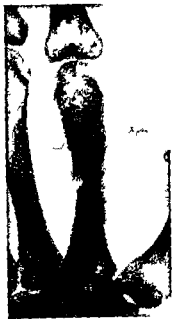


FIG. 368.—Syphilitic osteitis may cause a fracture. The patient was aged 38 years. Clinically the condition was mistaken for tuberculosis.



FIG. 369.—Osteitis in tertiary syphilis. The alteration in cancellous structure is shown best in the distal end of the radius.

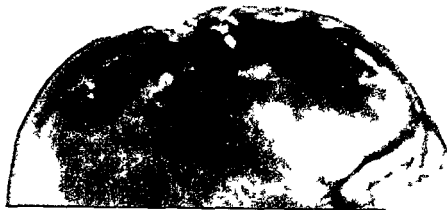


FIG 31 Extensive erosion of both tables of the skull in syphilis

liable to syphilitic involvement. Necrosis is often the predominant feature

Destruction of facial bones and invasion of the cranial vault are frequently followed by secondary infection and the formation of sequestra. Involvement of both tables of the skull may serve to differentiate this condition from Paget's disease (Fig 371)

THE LOCALISED LESIONS OF GUMMATA are often cortical (Fig 372) but may be central (Fig 373). They appear as hyperplastic periosteal nodes which are more or less localised. The central gumma is usually associated with a peripheral zone of condensation and subperiosteal bone formation. The latter consolidates and causes a localised thickening of the shaft. Both varieties tend to break down in the centre without the formation of sequestra. The central



FIG 37 —Osteitis of the clavicle and a cortical gumma of the humerus in an age 1 patient



gumma then appears as a comparatively large rarefied area surrounded by sclerosis and localised periostitis

The term *gummatous osteitis* is often loosely applied to a diffuse and massive osteitis of the shaft of a long bone



FIG. 373.—A central gumma with some periosteal reaction



FIG. 374.—Slight periostitis and syphilitic osteitis—the latter extending to the articular surface

## SYPHILIS OF JOINTS

**Congenital Disease**—The radiological examination of joints in congenital syphilis seldom contributes any useful information towards a diagnosis. The joint may be involved in osteochondritis and in late syphilis with hypertrophic or atrophic changes which are not diagnostic of the type of infection. *Clutton's joints* or bilateral symmetrical synovitis, are due to bursal or synovial involvement, and do not exhibit any characteristic radiographic appearances

A rare lesion in late congenital syphilis is a Charcot's joint, the diagnosis of which is described later

**Acquired Disease**—The joint lesions in the early stages of the acquired disease also lack characteristic features. There may be some widening of the joint space due to hydrops but this is similar to the hydrops from any other cause. In the tertiary stage, osteomyelitic or gummatous disease may extend to the end of the bone and cause joint changes (Figs 374 and 375). Sometimes both bones of the joint are affected, but the disease is often localised to one joint surface. The presence of multiple irregular areas of an osteoplastic type in the shaft with discrete areas of subchondral erosion, and the absence of general osteoporosis and haziness of bone should serve to differentiate the disease from tuberculosis. The clinical symptoms are in many cases similar to rheumatic disease, the differentiating radiological features of which are



FIG. 375.—Advanced osteitis with involvement of the joint surfaces. The radius appears to have escaped.

**Rheumatoid Arthritis**—Multiple joint involvement rarefaction of bone, marginal and later extensive erosions early loss of cartilage subluxation

**Osteoarthritis**—New bone formation in the form of osteophytes and loose fragments loss of cartilage

**Non specific Infective Arthritis**—Single joint, loss of cartilage, dense sclerosis combined with irregularity of articular surfaces

In addition all of these conditions are confined to the neighbourhood of the joint whereas the specific joint affection is due to a spread from pre-existing disease of the shaft

## CHARCOT'S JOINT

The Charcot's joint is an arthropathy due to a disease of the nervous system, which causes a neurotrophic disturbance with loss of the sensation of pain. It is most commonly found in adults suffering from tabes dorsalis or syringomyelia. Other rare causes are infantile tabes with congenital syphilis, paraplegia, peripheral nerve lesions spina bifida, leprosy, and traumatic injury to the spinal cord. Any joint may be affected. The most common sites



110 375 — A tal etio joint The patient was unaware of the condylar fracture of the femur It appeared to be of long duration and was imperfectly united



110 377 — A Claret joint with total luxation It began with atrophic changes but some loose bodies are now forming

are the knee ankle and foot hip spine and shoulder in decreasing order of frequency

The joints of the lower limbs are often affected in tabes. A secondary pyogenic infection may occur especially in the foot from a penetrating ulcer. The incidence of joint changes in this disease is greater in males than in females. Usually at least two thirds of the patients are males. The relative proportion is frequently higher than this but does not reach the figures for cord involvement which are approximately ten males to one female.

Of all patients suffering from syphilis approximately 2-4 per cent develop Charcot's joints. In syringomyelia the proportion is considerably higher probably over 10 per cent. In these patients the upper limbs are more frequently involved. The radiologist should not deviate from his diagnosis because the blood Wassermann is negative. This is to be expected in half the tabetic cases.

Two types have been described the atrophic and hypertrophic joint. The latter is more common.

### Radiological Features

The development of a Charcot's joint is usually progressive and insidious. Occasionally it is rapid especially after trauma of the joint. In the X ray department more



FIG 38—A Charcot's joint with subluxation and a great increase in density of the ends of the shafts. Eight years history of swelling of the knee.

patients are seen with the fully developed condition than with early manifestations.

**EARLY CASES**—The early features are excess of fluid in the joint, subluxation, sclerosis of articular surfaces and subchondral bone, irregularity of articular surfaces, varying width of the joint space, spontaneous fracture and multiple small sclerosed loose fragments of ill defined structure (Fig 376). In some patients the early changes are of an atrophic type which may alter in later years to the hypertrophic form.

**DEVELOPED CASES**—In the *atrophic* type there is a loss of substance at the

articular surfaces with a reduction in the size of the joint. A few small loose fragments of calcified material are sometimes present. The appearance may simulate a surgical excision of part of the bones (Fig. 377).

In the *hypertrophic* form the joint becomes disorganised. There is gross irregularity of the articular surfaces due to destruction and new bone formation. The latter may be in the form of osteophytes or loose fragments within or without the joint. The contour of the joint is both irregular and larger than normal. The presence of fluid laxity of ligaments and destruction of articular surfaces leads to subluxation or dislocation. The articular surfaces and excess bone are characterised by an increased density and the final result can seldom be mistaken for any other condition (Fig. 378).

Patients who have developed a Charcot's joint often show early radiographic changes in other joints which are symptom free. Early changes in the spine appear as an increase in density of the bodies with thinning and irregularity of the disc spaces. This density may affect only the opposing surfaces but it eventually extends throughout the whole body which becomes larger than normal. New bone formation takes place at an early stage and later there develop the same characteristic features of destruction and excess of new bone.

There are no radiographic features by which a Charcot's joint of syringomyelia may be distinguished from that of tabes. As previously stated the distribution in syringomyelia is usually in the upper limbs as the dissociated anaesthesia affects these segments (Fig. 379).



FIG. 379. A Charcot's joint in syringomyelia.

# REFERENCES

- STOKES J. H. *Modern Clinical Syphilis*. W. B. Saunders Co., Phil. 1934, 649.  
 WEGNER C. *Lirchows Arch. f. Path. Anat.* 1870, I, 305.  
 WILE J. and BUTLER M. G. *Journ. Amer. Med. Assn.* 1930, XCIV, 1003.

## CHAPTER XXXI

### UNCOMMON INFLAMMATORY DISEASES OF BONE

BY

E ROHAN WILLIAMS MD FRCP FFR DURE

#### ECHINOCOCCUS (HYDATID) DISEASE OF BONE

HYDATID DISEASE is decidedly uncommon in England as the skeleton is involved in only 1 per cent of all cases it will be realised that echinococcus of bone is rare. In a review of 244 cases showing bone involvement *Bauer* found that the pelvic bones were affected in 30 per cent the vertebrae in 17 per cent the humerus in 14 per cent the tibia in 12 per cent and the femur in 10 per cent.

The radiographic appearances will depend upon the presence or absence of complications. The disease usually begins in the cancellous bone osteolysis is predominant and slowly extends towards the cortex (Fig 380). The lesion in bone does not retain the cystic characters that are seen in the liver or lungs and the margins of the osteolytic area are very irregular giving an impression of multiloculation. When the cortex is involved a slight periosteal reaction may result and pathological fractures occur. At this stage the radiographic appearance is that of a coarse crumpled honeycomb (*Claessen*).

Secondary infection will follow ulceration of the overlying skin and sequestra may form occasionally the cyst walls may be seen to be calcified within the bone.

Other than this coarse honeycomb appearance there are no characteristic radiographic features. A differential diagnosis has to be made from osteogenic sarcoma giant cell tumour and fibro cystic disease in the long bones and from carcinomatous metastases and tuberculous caries in the spine.

A final diagnosis will only be realised from biopsy or from the demonstration of echinococcus lesions in the lungs or liver.

#### YAWS IN BONE

In yaws approximately one fifth of all cases in the tertiary stage show skeletal lesions. *Maul* after a thorough investigation in 1919 came to the conclusion that in early bone involvement certain changes occurred which were peculiar to yaws. *Maul* described

(1) Multiple osteolytic oval areas in the long bones especially in the radius ulna and tibia the long axes of these areas lay in the long axes of the bones



FIG 380—Hydatid disease in the tibia  
a multilocular but uncomplicated lesion  
(From a specimen in the Museum St Mary's Hospital London. UJ kind permission of Dr W D Veacock.)



FIG 381—Yaws lesions in the tibia. AP and lateral views of the same specimen  
(From the I G S Museum No 33721, bJ kind permission of the Curator.)

and measured 2–3 cm. The areas were in cancellous and cortical bone, and there was practically no neighbouring periosteal new bone formation (Fig 381).

(ii) Similar zones in the articular surface, especially where these surfaces were somewhat exposed to trauma, e.g. in the femoral condyles.

(iii) Somewhat uncommonly superficial cortical lesions which provoked a considerable localised nodular periosteal new bone formation.

The response to arsenical therapy in such lesions is very rapid and complete healing occurs. In the chronic stages the typical appearances described are lost and increasing new bone formation of an irregular type occurs so that a differentiation from syphilitic osteoperiostitis is practically impossible. The sabre tibia may therefore be seen in yaws as well as in tertiary syphilis. *Lawford Knaggs* is of the opinion that the earlier changes are not simulated by any other bone lesion.

### LEPROSY IN BONE

Bone lesions in leprosy bear no constant relationship either to the degree or to the type of clinical involvement. Changes occur in both the cutaneous and in the nerve forms and are usually more marked in the latter.

The changes may usually be regarded as neurotrophic i.e. secondary to the nerve lesions rather than as a direct leprous osteitis. Bone changes of a minor type may improve and regress with variations seen clinically in the soft parts.

*Murdock and Haller* have made an extensive study of the skeleton in lepers. They investigated 140 unselected cases and found bone lesions in 108 cases (77 per cent). Of these the majority showed changes in the small bones of the hands and feet the long bones rarely presenting any abnormality.

Leprous bone lesions are osteolytic unless complicated by a secondary pyococcal infection rarely in severe degrees of cutaneous nodular involvement underlying bones may show an appreciable periosteal reaction.

*Murdock and Haller* classify the changes they observed thus

(i) LOCALISED BONE DEFECTS—These were especially seen in the terminal phalanges. In the subungual cancellous tufts small areas of bone destruction appeared as nicks slices and frayings. The whole tuft may be destroyed leaving the rest of the phalanx with a collar stud appearance.

(ii) GROSS BONE DEFECTS—Regional atrophy followed by destructive osteitis and osteomyelitis were seen. Sometimes marked cystic degeneration was noted (*leprous osteitis multiplex cystica*).

(iii) ARTHRITIC CHANGES—These may be both atrophic and hypertrophic in character.

In children the bone changes were most marked at the age of epiphyseal fusion.

The radiographic differential diagnosis has to be made from the changes seen in syringomyelia tabes dorsalis angitis obliterans Boeck's sarcoid and tuberculous osteitis multiplex cystica.

### MALTA (UNDULANT) FEVER

There are very few reports in the literature of radiological findings in skeletal involvement by the *Brucella melitensis*. *O'Donoghue* has reported a case of acute septic arthritis due to this organism but no peculiar changes were noted. *Kulowski and Link* suggest that if the spine was radiographed more



often in cases of Malta fever which have severe back pain, then bone changes would be seen more frequently. In their own case, marked destructive changes occurred at the inter articular facets in the lower lumbar spine, the radiograms also showed a typical increase in the psoas muscle shadow, due to the presence of a psoas abscess. No changes were found in the vertebral bodies.

### ACTINOMYCOSIS OF BONE

Skeletal actinomycosis usually occurs as a result of an extension from neighbouring soft part actinomycotic foci, hence the sites usually affected are

- (i) The pelvis and lumbar spine from ileo caecal foci
- (ii) The ribs and dorsal spine from pleuro pulmonary foci

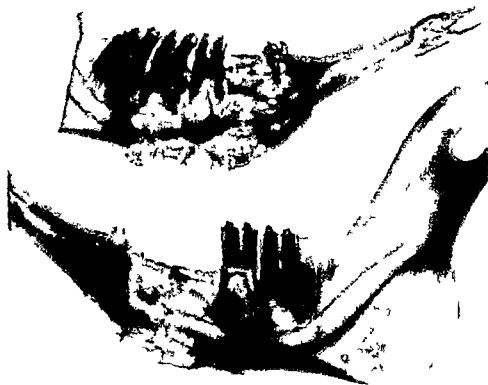


FIG. 382.—Actinomycosis of the mandible in cattle: two specimens of 'lumpy jaw'. From the Museum, St. Mary's Hospital, London, by kind permission of Dr. W. D. Newcomb.

(iii) The mandible from soft part involvement in the mouth and cervical lymph nodes.

Occasionally metastatic bone actinomycosis occurs in situations far distant from the primary focus, which is usually pulmonary: such cases have been reported by Krogius, and by Allenbach, Sartori, and Zimmer.

In the commoner type of bone involvement, the radiographic appearances

are those of a ragged irregular osteolytic process new bone formation is only slight unless gross secondary infection supervenes In the lumpy jaw of cattle however new bone formation may be very gross (Fig 382)

The differential diagnosis in the extension form has to be made from chronic osteomyelitis and from tuberculous caries There are no pathognomonic radiographic appearances and although the nature of the lesion may be suspected the proof rests with the demonstration of the fungus *Tabb and Tucker* have made a special study of vertebral actinomycosis and state that the lesion usually involves two or more vertebrae that there is no predilection for the bodies but that pedicles and laminae are equally affected that collapse and disc destruction do not occur that sequestration is seldom observed but abscess formation is usual and that the osteolytic areas are sharply defined with normal bone intervening

Rarely a mandibular focus may be primary the entrance of the fungus being made through the root canals of carious teeth When such a focus is noted differential diagnosis from neoplastic and cystic lesions and from simple infective apical granulomata will have to be considered

### BLASTOMYCOSIS AND COCCIDIOIDAL GRANULOMA OF BONE

Blastomycosis may involve the skeleton in systemic blastomycosis or rarely as a result of direct spread from cutaneous lesions of the forearm and hand In the direct spread form the radiographic appearance will be that of slight bone destruction with neighbouring sclerosis In systemic blastomycosis the lesions usually occur in cancellous bone bone destruction predominates with abscess cavitation new bone formation is slight

The lesions are usually multiple and appear to have a predilection for bony prominences e.g. the malleoli the tibial tuberosities Joint involvement is usually late The vertebrae may be affected and destruction extends throughout all vertebral components and to adjacent ribs

In the tubular bones of the hands and feet periosteal reaction may be prominent and the dactylitis closely resembles that due to either tuberculosis or syphilis

*Kypins* has reviewed the literature and has reported three cases in detail and *Carter* has made an extensive radiological study of the fungus infections of the skeleton *Carter* concludes that the mycotic lesions resemble each other more closely than they resemble tuberculous syphilitic or chronic osteomyelitic lesions but that the mycoses in bone have few pathognomonic features In regions where the mycoses are endemic the following features should suggest that a pathological fungus is responsible

(i) Multiple lesions (ii) cancellous bone selection (iii) invasive character (iv) punched out foci in the calvarium (v) vertebral involvement without limitation to the bodies and (vi) involvement of the thoracic parietes from within the thorax

## SPOROTRICHOSIS

Sporotrichosis in bones is very rare, but does occur in the hands and feet as a result of direct spread from cutaneous lesions. Isolated cases have been reported in which sporotrichosis metastasised to bone. A striking example of this rare type has been published by *Altschul*.

The radiographic appearances are those of a chronic infective osteoperiostitis, with bone destruction, new bone formation, and sequestration. The appearances may be very similar to those seen in a secondary infected bone tuberculosis.

## MYCETOMA PEDIS (MADURA FOOT)

The responsible organism in Madura Foot is a mycetoma fungus resembling actinomyces, but found only in tropical zones. *Benassi* and *Fiaschi* found the radiographic changes to be those of osteoperiostitis, with predominating new bone formation. There were no characteristic features.

## BIBLIOGRAPHY

## HYDATID DISEASE

- BAUER B., *Fortschr ad Geb d Rontgenstr*, 1913, **XIX**, 285  
 CLAESSENS, G., *Acta Radiologica, Suppl* 11, 1928  
 COLEY, B. L., *Journ Bone and Joint Surg*, 1932, **XIV**, 577  
 DEW, H. R., *Hydatid Disease*. Australian Medical Publishing Co. Ltd. Sydney, 1928

## YAWS

- KNAGGS R. L., *Inflammatory and Toxic Diseases of Bone*. John Wright, Bristol 1926  
 MACL, H. G., *Amer Journ Roentgen*, 1919, **VI**, 421  
 WILSON, P. W., and MATHIS, M. S., *Journ Amer Med Assn* 1930, **XCIV**, 1289

## LEPROSY

- MURDOCK, J. R., and HUTTER, H. J., *Amer Journ Roentgen*, 1932, **XXVIII**, 598

## MALTA FEVER

- KULOWSKI, J. and VINKE, T. H., *Journ Amer Med Assn*, 1932, **XCIX**, 1656  
 O'DONOGHUE A. F., *Journ Bone and Joint Surg*, 1933, **XV**, 506

## ACTINOMYCOSIS

- ALLENBACH, E., *et alia*, *Rei d'Orthop*, 1931, **XVIII**, 572  
 KROGUS, A., *Acta Chir Scandinav*, 1928, **LXIII**, 121  
 PARKER C. A., *Journ Bone and Joint Surg*, 1923, **V**, 759  
 SIMPSON, W. M., and MCINTOSH, C. A., *Arch Surg*, 1927, **XIV**, 1166  
 TABB, J. L., and TUCKER J. D., *Amer Journ Roentgen*, 1933, **XXIX**, 633

## BLASTOMYCOSIS AND COCCIDIOIDAL GRANULOMA

- CARTER R. A., *Radiology*, 1934, **XXIII**, 1  
 RYFINS, E. L., *Radiology* 1934, **XXII**, 77  
 SUTEJEV, G. *et alia*, *Fortschr ad Geb d Rontgenstr*, 1930, **XL**, 475  
 TAYLOR, R. G., *Amer Journ Roentgen*, 1923, **VIII**, 531

## SPOROTRICHOSIS

- ALTSCHUL, W., *Amer Journ Roentgen*, 1926, **XV**, 3

## MADURA FOOT

- BENASSI E., and FIASCHI, P., *Chir Org Moen*, 1929, **XV**, 356

## CHAPTER XXVII

### ACUTE AND CHRONIC ARTHRITIS

BY

E. ROHAN WILLIAMS, M.D., F.R.C.P., F.R.D.V.R.

#### ACUTE ARTHRITIS

ACUTE ARTHRITIS may be either non suppurative or suppurative.

Non suppurative Acute Arthritis may occur as a result of trauma to a joint and during the course of acute rheumatic fever and certain acute specific fevers. The radiographic appearances of an affected joint are not striking. Those seen in *acute rheumatic arthritis* may be considered as typical of the group. The departures from normal findings are



FIG. 383.—Subacute gonococcal arthritis of the knee joint. Note the subarticular translucent zone and marked loss of bone (osteolysis).

(i) Slight increase in joint space (ii) slight lack of sharpness in bone detail owing to (iii) synovial effusion and peri articular swelling. There is little or no disturbance of calcium distribution except as a result of disuse osteoporosis. The articular cartilage is not destroyed and thus no underlying bone changes occur. The recovery, judged by radiographic observation, is usually complete although contractures may occur in the peri articular tissues.

**Suppurative Acute Arthritis**—This may result from the infection of a



FIG. 384.—Acute pneumococcal arthritis of the right hip joint secondary to a pneumococcal empyema. Note the loss of joint space and slight bone destruction at the articular margins.

joint by the staphylococcus streptococcus gonococcus and pneumococcus and may occur during the course of certain specific fevers.

The radiographic changes depend not so much on the type of the organism as on its virulence and on the resistance offered by the subject.

The changes will be the least when the effusion is serous, more marked if seropurulent and gross if purulent.

**RADIOGRAPHIC APPEARANCES**—The earlier changes are not striking and hence may easily be overlooked. They are

(i) Increase in joint space

(ii) Osteoporosis in the neighbouring bones this may be very marked

(iii) Loss of bone detail, this is especially apparent in the subarticular areas. Sometimes the articular cortex appears to be relatively dense, though poor in detail, but in a linear zone beneath the cortex, osteoporosis and loss of detail may be very marked. This subcortical translucent zone is especially striking in certain cases of gonococcal arthritis before any actual cartilage

destruction has occurred, this appearance, however cannot be considered as peculiar to a gonococcal infection (Fig 383)

When cartilage destruction has taken place, the radiographic features are more striking. The following changes will be noted

(i) Reduction of joint space, which will be most marked at opposing joint surfaces especially when these are responsible for pressure transference (Fig 384)

(ii) Marginal bone destruction, again most apparent where cartilage loss is severe (Fig 385)

(iii) Massive necrosis with sequestration. This may be seen when femoral head separation occurs in acute arthritis of the hip joint

Pathological dislocation complete or incom-



FIG 383.—Acute arthritis of the hip joint due to a streptococcal (hemolytic) infection.

plete may occur before or after cartilage destruction

In the recovery phases calcium distribution returns to normal except at the actual eroded margins, which show an irregular sclerosis or undergo bony ankylosis with opposing surfaces (Fig 386)

### CHRONIC ARTHRITIS

It is not proposed in this section to enter into a profound study of the perplexing problems of the aetiology and pathology of the chronic arthritides

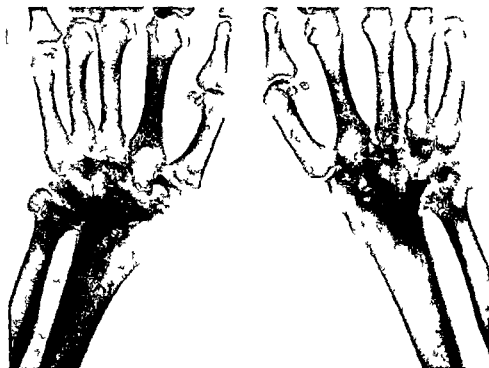


FIG. 386.—Final result of an old bilateral gonococcal arthritis involving wrist and carpal joints with ankylosis.

It is intended that the general conclusions reported by the Arthritis Committee of the British Medical Association in 1933 be accepted for the most part and that concentration in this section will be devoted to radiological description of the findings in chronic arthritic diseases.

**Classification**—The B M A Arthritis Committee adopted the following classification

RHEUMATOID ARTHRITIS	{	Chronic Polyarthritis
Synonymous with		Atrophic Arthritis
		Proliferative Arthritis
CHRONIC VILLOUS ARTHRITIS		
OSTEO ARTHRITIS	{	Hypertrophic Arthritis
Synonymous with		Degenerative Arthritis
		Osteo arthrosis
SPONDYLITIS		
(a) Osteo arthritica		
(b) Ankylopoietica		

This classification will be adhered to in this section. Spondylitis receives especial consideration.

## RHEUMATOID ARTHRITIS

**General Observations**—The most suitable term in radiological descriptive writing is atrophic the term proliferative used widely in America is fully justified on morbid anatomical grounds but if used in radiology tends to cause confusion with the hypertrophic changes of osteoarthritis

The B V A Arthritis Committee recognised (i) *Primary Rheumatoid Arthritis* and (ii) *Secondary Rheumatoid Arthritis* The primary group as its name implies is that in which no cause can be found for the arthritis the secondary group is that in which a focal or general infection is held responsible

**Clinical Features**

**PRIMARY TYPE**—Women are affected almost exclusively the age incidence is from 20 to 45 years Prodromal symptoms of peripheral wasting and vasomotor disturbances are not uncommon The onset may be acute or subacute and the arthritis usually begins in the small joints of the hands about which there is a characteristic fusiform swelling associated with marked muscular wasting As the condition advances contractures and deformities result and fibrous and bony ankylosis may occur General health is adversely

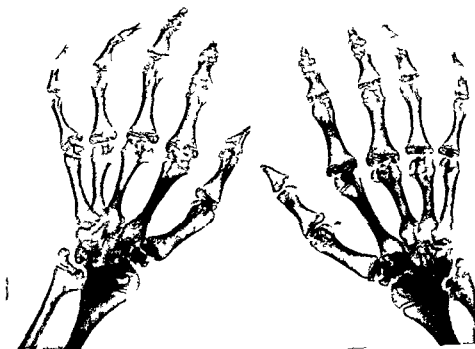


FIG 38 The hand in Still's disease quiescent phase Same case as Fig 388



affected, and anemia may be prominent. The disease takes a very chronic course, and remission and exacerbation periods are common.

The *ætiology* is quite uncertain. The definition of this group is such that no known septic focus can be held responsible. The autonomic nervous disturbances at least suggest that some endocrine imbalance may be present.

**SECONDARY TYPE**—Males and females are affected in equal numbers. Most cases show an onset between 20 and 40 years, but children and elderly subjects may be affected. The onset may be associated with an acute febrile



FIG. 388.—The hip joints in Still's disease—quietest phase. Same case as Fig. 387.

illness closely resembling acute rheumatic fever, or may be subacute and showing a localisation of signs to the joints. The condition in children is known as *Still's disease* (Figs. 387 and 388).

In both forms there is a polyarticular and usually symmetrical joint involvement, the more peripheral joints are most commonly affected, but the ankles, knees, elbows, wrists, and spinal joints are by no means exempt.

The course will depend on the treatment directed at the responsible septic focus. If this is adequate complete recovery may result, if inadequate then the subacute process will become chronic with occasional intermissions. Vasomotor disturbances, secondary anaemia, and myocardial degeneration are occasional accompaniments.

### Radiographic Appearances

The primary and secondary types of rheumatoid arthritis both show similar radiographic changes and thus a general description of both types will be given without differentiation.

Scott has pointed out the importance of a standardisation of technique all too commonly radiography in chronic arthritis is haphazard and consequently radiological diagnosis is inaccurate and early changes are frequently overlooked.

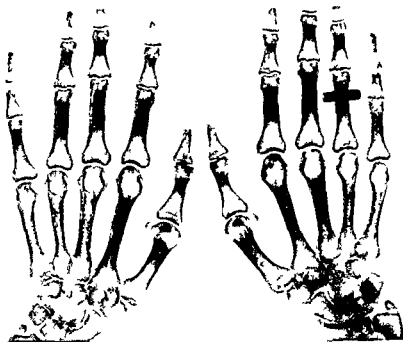


FIG. 389 Active rheumatoid arthritis of hands early stage

**EARLY STAGE**—In the earlier stages of rheumatoid arthritis the following changes will be noted

(i) **Osteoporosis** this is both *generalised* affecting the whole skeleton and *localised* in the bones in the vicinity of the affected joints. Scott describes an alteration of the bone density balance in which a bone will show an almost uniform density in lieu of the normal variations of density in (a) the cortex (b) the shaft cancellous bone (c) the cancellous bone of the extremities.

(ii) Slight increase in joint space followed by loss of joint space as the cartilage is destroyed.

(iii) Fusiform soft part swelling about the joints (Fig. 389).

**LATE STAGE**—The osteoporosis and loss of joint space will become more marked and in the later stages the following changes are seen

(i) Extreme osteoporosis

(ii) Subluxation and full dislocation of joints may occur

(iii) Joint space loss is complete and bony ankylosis will follow. When the disease becomes quiescent the bone density balance returns towards the normal, the osteoporosis generally is less apparent but deformities remain.

*In the hands* so commonly involved a characteristic appearance will be appreciated.

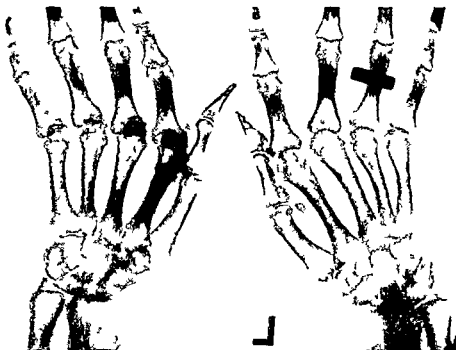


FIG. 330 Active rheumatoid arthritis of hand—advanced. Note the metacarpophalangeal subluxations and the ulnar deviation of the fingers.

(i) The metacarpophalangeal joints, proximal interphalangeal joints and the opposing articular surfaces of the scaphoid and radius are especially affected.

(ii) An ulnar deviation of the fingers at the metacarpophalangeal joints is characteristic, resulting from soft part contractures and from medial subluxation of the proximal phalanges on the metacarpal heads (Figs. 330 and 331).

### CHRONIC VILLOUS ARTHRITIS

Chronic villous arthritis occurs in women at the climacteric and is often associated with a marked increase in weight. No pathological bone changes are seen in a radiogram unless the condition is of long standing, when simple osteoarthritic changes will usually be appreciated.

## OSTEO-ARTHRITIS

Osteo arthritis is the term applied to that disease, or group of diseases characterised by degenerative changes in the articular cartilages, followed by hypertrophic new bone formation. Broadly speaking osteo arthritis may be regarded as *primary* when there is no outstanding ætiological association, and *secondary* when a joint has been subjected to a severe single injury or to

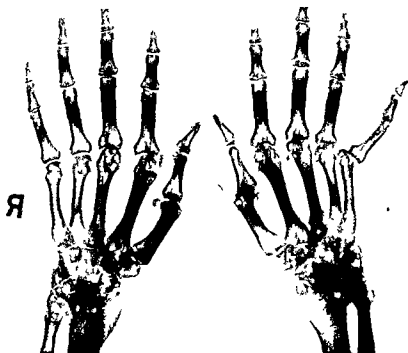


FIG. 391.—Chronic arthritis of hand is quiescent. Secondary rheumatoid arthritis with multiple irregular defects in juxta-articular bone resembling chronic gouty arthritis.

minor repeated injuries to abnormal stress and strain or has been the site of some inflammatory or deforming process.

Using this terminology, primary osteo arthritis may be regarded as a disease of subjects of late middle age or of advanced years. The general health is usually good although other degenerative changes e.g. arterio-sclerosis, may be present.

The larger joints are most commonly affected, but no joint is exempt. The clinical onset is slow and the early complaints are those of stiffness and aching. Pain will be complained of when cartilage destruction is appreciable. The degree of clinical severity cannot be judged from the degree of hypertrophic new bone formation.

**Radiographic Appearances**—It should be realised that in different cases



FIG 113—Early osteo arthritis of the h p joint, showing pseudo cystic changes in the roof



FIG 102—Early osteo arthritis changes in the knee joint

the various changes enumerated below will vary considerably i.e. in a given case cartilage destruction may be severe whilst new bone formation may be slight. The converse may also hold.

**EARLIER CHANGES**—(i) The articular marginal angles become sharper and more acute than normal. spur formation and lipping results from new bone formation. opposing spurs tend to approach one another (Fig. 392).

(ii) The joint space becomes narrower than normally owing to the destruction of articular cartilage.

(iii) Sclerosis of the articular margins takes place.

(iv) Small translucent zones will be seen in the bone beneath the articular margins. these may be rounded and due to degenerative pseudo cyst formation or somewhat irregular resulting from fibrous tissue replacement of bone (Fig. 393).

**LATER CHANGES**—(i) Increasing spur formation (Fig. 394).

(ii) Marled cartilage destruction.

(iii) Increasing sclerosis of the articular margins with a loss of their normal curves. this is especially marked at pressure points. The sclerosis may extend through moderately wide areas in the neighbourhood of a joint.

(iv) Tendency to subluxation.

(v) Separation of osteophytic spurs to form fringe osteophytes or loose bodies within the joint.

### OSTEO-ARTHRITIS IN CERTAIN JOINTS

**Hip Joint (Morbus Coxæ Senilis)**—The pseudo cystic areas are often well seen in the roof of the acetabulum which may also become intensely sclerosed. New bone formation is often prominent and is seen chiefly at (i) the outer superior margin of the acetabulum which may overhang the femoral head or at (ii) the lower part of the acetabular floor. when this is gross the femoral head will be pushed upwards and outwards (Fig. 395).

Occasionally osteo arthritic *protrusion of the acetabulum* may be seen. The acetabular cavity deepens and the femoral head becomes enveloped. cartilage erosion adds to the depth of the socket. Eventually the inner wall of the acetabulum may bulge into the true pelvic cavity (Fig. 396). This deformity was first described by *Otto* and has been fully studied by *Valentin* and *Muller*. It may occur in conditions other than osteo arthritis.

**Shoulder Joint**—In contrast to the radiographic appearances of osteo arthritis of the hip the same disease in the shoulder only produces slight departures from normal. New bone formation is minimal. marginal sclerosis of the glenoid rim is usually the only gross change (Fig. 397). The acromio clavicular joint often shows more typical changes when the shoulder joint clinically involved shows little or no radiographic pathology.

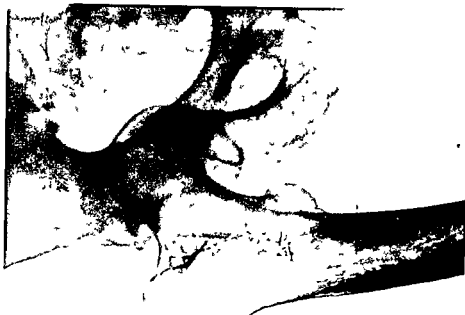
**Interphalangeal Joints of the Hands**—Apart from primary cases *Scott* has shown that osteo arthritis of the hands is frequently observed in manual



110 305—A view of the front of the face of the person at the top of the page. The person is wearing a dark, possibly fur-lined garment. The image is heavily stylized, with deep blacks and bright whites, giving it a graphic, almost abstract quality. The person's features are partially obscured by shadows and the texture of the garment.



311—A view of the front of the face of the person at the top of the page. The person is wearing a dark, possibly fur-lined garment. The image is heavily stylized, with deep blacks and bright whites, giving it a graphic, almost abstract quality. The person's features are partially obscured by shadows and the texture of the garment.



116 300—Osteoarthritic protrusion of the acetabulum  
(Osteoarthritis)



116 397—Osteoarthritic protrusion of the acetabulum  
(Osteoarthritis)



workers in certain trades and that different occupations show different interphalangeal joint distributions

The terminal and middle interphalangeal joints are usually affected in contrast to the osteoporosis of rheumatoid arthritis there is no appreciable alteration of calcium content nor of the bone density balance Following cartilage destruction subluxation is especially liable to occur (Fig. 398)

Three types of bone change may be seen to account for the marked localised swelling about the affected joints These are

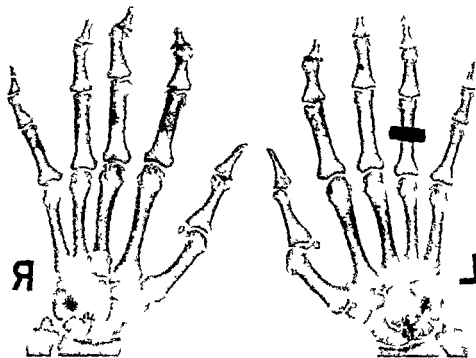


FIG. 398.—Osteoarthritis of the interphalangeal joints. Note the absence of osteoporosis.

(i) Sharp protruding *osteophytic spurs*—the new bone formation is distinct from the original articular margin

(ii) Prominent splaying out of the bases of the phalanges—the false spurs being continuous with the original margin (*Heberden's node*)

(iii) *Small Peri-articular Ossicles*—these are dissociated from spur formation and are usually smooth and rounded—they lie opposite the joint level

**Clinical Significance of Radiographic Changes**—The degree of cartilage erosion is much more significant than the degree of new bone formation. Extreme hypertrophic bone changes are often discovered incidentally in a radiographic examination without any clinical complaint which can be attributed to the osteoarthritis. It is the duty of the radiologist to note such

changes but he is seldom justified in assuming that spur formation will alone account for the clinical findings. In short advanced hypertrophic changes must not preclude a careful search for other pathological changes of more serious significance.

### CHRONIC SPONDYLITIS

Chronic arthritis of the vertebral column may be classified in the following manner

(i) *Rheumatoid Spondylitis*—spinal involvement in rheumatoid arthritis

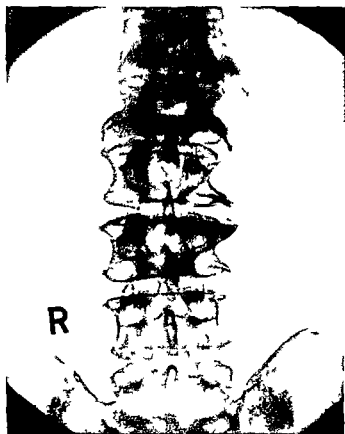


FIG. 399. Spondylitis osteoarthritis.

(ii) *Chronic Infective Spondylitis* usually the chronic stages of an acute infection of the vertebral column

(iii) *Spondylitis Osteoarthritis*

(iv) *Spondylitis Ankylopoietica*

In the first two types the vertebral changes do not differ from arthritic processes in other joints of the same origin.

*Spondylitis Osteoarthritis* in no way differs from osteoarthritis in other joints; it must be remembered that the disease may affect the small joints between the articular processes as well as the body margins. Hypertrophic spur formation is sometimes very gross and opposing

spurs may meet but never actually fuse (Fig. 399).

Secondary osteoarthritis spondylitis is frequently seen in a localised form in association with (i) curvature deformities of the spine (ii) old compression body injuries (iii) healed tuberculous caries osteochondritis nucleus pulposus prolapse.

*Spondylitis Ankylopoietica*—There is very little conformity of opinion as

to the significant factors in the production of ankylosing spondylitis. The B.M.A. Arthritis Committee criticises the separation of ankylosing spondylitis into the two forms—

(i) Spondylose heredo traumatique (ii) Spondylose rhizo melique—of the French writers is being untenable on aetiological and clinical grounds. Nevertheless although these titles may give a false impression there is no doubt that the consideration of at least two forms of ankylosing spondylitis is fully justified by commonplace radiological observation and by such careful morbid anatomical observations as those of *Lauford Knaggs*. The descriptions given by *Marie* and *Strumpell* and by *Bechterew* are still acceptable although the quasi aetiological titles of the two forms may not be

*Knaggs* recognises

(i) *Spondylitis Ossificans Ligamentosa* (the *Marie Strumpell* type spondylose rhizo melique)

(ii) *Spondylitis Muscularis* (the *Bechterew* type spondylose heredo traumatique)

**SPONDYLITIS OSSIFICANS LIGAMENTOSA**—This condition affects young male subjects almost exclusively. It is probably rather of an inflammatory than of a degenerative nature a few cases have been reported which were almost certainly of gonococcal origin.

The most important feature is ligamentous ossification. The anterior common ligament is most frequently affected in the early stages but as the disease advances the posterior common ligaments, ligamenta subflava, the capsular ligaments, the costovertebral ligaments and the interspinous ligaments are all involved. In certain cases in which the vertebrae show these changes the larger proximal limb joints show an ankylosing arthritis in which the ankylosis is a direct bone fusion and not a ligamentous ossification.



FIG. 4. Spondylitis ankylopoietica bamboo-pneumolitos. Spondylitis ossificans ligamentosa. Note the sacro-lac obliteration and the preservation of the intervertebral disc space.

The sacro iliac joints are almost invariably affected in the earliest stages

**Radiographic Appearances**—The process usually begins in the sacro iliac joints and lower lumbar spine moderate osteoporosis is seen, followed by a slow obliteration of the sacro iliac joint space by new bone, in lateral



FIG. 401. Dorsal kyphosis in an elderly subject the disc atrophy is pronounced the upper vertebrae show early ankylosis of the type seen in spondylitis muscularis atill the lower vertebrae show the commoner osteoarthritic changes.

views of the lower lumbar vertebrae the anterior common ligament shows some bone deposition bridging the gap anteriorly across the disc space as the condition advances the whole gap between the body margins is covered by ligamentous ossification but the disc itself is not encroached upon (Fig. 400)

The process usually advances in a caudocranial direction and eventually many vertebrae will be completely enveloped in ossified ligaments in an antero posterior view the description of bamboo spine is very apt. The osteoporosis will be less obvious as the ankylosis increases.

It is interesting to note that certain authors (*Oppel Hall*) regard ankylosing spondylitis of this type as a possible manifestation of parathyroidism the removal of calcium from the bones and its deposition in the ligaments, together with clinical improvement and arrest of the disease after parathyroidectomy lends support to this view. Certain cases show an increase of serum calcium content.

**SPONDYLITIS MUSCULAPIS**—This form is usually found in elderly subjects in the upper dorsal and lower cervical spine.

*Knaggs* believes that a muscular atony is primarily responsible, this

leads to a secondary disc atrophy, which is the most important radiographic feature the disc space narrows especially anteriorly, and the body margins impinge upon one another and eventually fuse directly (Fig. 401). Slight ligamentous ossification may be present but it is not the essential feature

In most cases, the body fusion is restricted to the anterior regions, but in long standing cases, the fusion may be complete between the bodies

Joints elsewhere in the body are not affected

## BIBLIOGRAPHY

- ' Arthritis Committee, BMA Report,' *Brit Med Journ* , 1933 (June 17th), 1033.  
 BECHTEREW, W , *Neurolog Centralbl* , 1893 **XIII** 425  
 HALL E W , *Amer Journ Roentgen* , 1933 **XXA**, 608  
 KNAGGS, R L , (1) *Brit Journ Surg* 1925, **XII**, 524 , (2) *Inflammatory and Toxic Diseases of Bone* John Wright Bristol 1926  
 MARIE, P , *Pet de med* , 1898 **XVIII**, 285  
 OPPEL, V A , *Ann Surg* 1929 **XC** 978  
 SCOTT, S G (1) *Chronic Rheumatic Arthritis (The Hand)* Oxford University Press London 1935 , (2) *Brit Med Journ* , 1932 (February 13th) 275  
 VALENTIN, B , and MÜLLER, H , *Arch f Klin Chir* 1921, (**CVII**) 523

# PART THREE

## SECTION VI

### OSTEOCHONDritis

BY  
E. ROHAN WILLIAMS M.D. F.R.C.P. F.F.R. D.M.R.E.

#### CHAPTER XXXIII

#### INTRODUCTION

BEFORE THE advent of radiology several observers had noted that pathological conditions bearing a superficial resemblance to tuberculous lesions were not uncommon in epiphyseal zones in children the clinical course was much milder and the prognosis better than in the tuberculous processes.

Radiological studies have proved an ideal method of investigating the living pathology in this group of non tuberculous necroses of growing bone

#### OSTEOCHONDritis

	PRIMARY CENTRES	SECONDARY CENTRES
SPINE	Vertebral Body (Calve 1905)	Epiphyseal Plates (Steinmann 1911)
UPPER LIMB	Adult Semilunar (Klenbock 1910) Scaploid (Frederick)	Claicle Sternal (Friedrich 1904) Humerus Head (Lewin 1904) Humerus Capitulum } (Brailford 1930) Radius Head Ulna Distal (Burn 1931) Metacarpal Heads (Maclaure 1904)
LOWER LIMB	Patella (Kohler 1908)  Atraculus (Mouchet 1908) Scaphoid (Kohler 1908) Medial Cuneiform (Buschke 1934) Adult Scaphoid (Brailford 1930)	Iliac Crests (Bulman 1904) Pubic Symphy (Van Neck 1904) Ischopubic Junction (Vollant 1904) Acetabulum (Brailford 1930) Femur Head (Perthes—Legg—Calve 1916) Femur Neck (Gutge and Hertzog 1931) Femur Trochanters (Monde Felix 1904) Patella Polar (Sinding Larsen 1911) Tibia Head (Ritter 1909) Tibia Tubercle (Osgood 1903) Os Calcis (Sever 1910) First Metatarsal Proximal (Wagner 1930) Second and Third Metatarsal Heads (Frederick 1914)

Numerous observers have described such cases affecting different anatomical zones unfortunately it became customary to speak of the condition in each zone by the name of the original observer. This form of nomenclature has the disadvantage that it gives no information as to the underlying pathological process further it has marked for many years the close relationship of the lesions in the different affected anatomical sites. The accompanying table (p. 474) shows the different areas in which the lesion has been noted together with the name of the original author and the year of the observation report.

During the last few years there has been a strong movement on the part of such authoritative writers as *Axhausen*, *Harbin* and *Brailsford* to group these conditions together as representing a similar pathological process in different zones. *Axhausen* uses the term necrosis of epiphyses and short bones. *Harbin* calls the condition osteochondritis of the growth centres and *Brailsford* used the simplest term osteochondritis.

In this section it is proposed to accept the name osteochondritis. A justifiable criticism of the term is that it suggests an inflammatory lesion which is very unlikely. Osteochondritis is widely accepted in England and until our knowledge of the aetiology and pathology is more complete it is felt that no better term can be put forward.

Osteochondritis most commonly occurs in juvenile subjects however in certain bones in adults changes sometimes occur which very closely resemble those seen in the juvenile cases. It is not proposed in this section to give an account of osteochondritis in adult bones in which the association of trauma is so close that it has been considered wiser to consider these as examples of post-traumatic malacia and they are consequently dealt with in the section on injuries of bones and joints.

The adult bones which may be affected in this form of osteochondritis are the carpal scaphoid (*Preiser*) semilunar (*Kienbock*) vertebral body (*Kummel*) tarsal scaphoid (*Brailsford*) neural arch of the fifth lumbar vertebra in spondylolisthesis (*Brailsford*) and the great toe sesamoids (*Renanier*).

Juvenile osteochondritis may occur in primary ossific centres (e.g. in the tarsal scaphoid) or in secondary ossific centres (e.g. in the calcaneal epiphysis).

### ÆTIOLOGY

There is no general agreement concerning the aetiology of osteochondritis. Several theories have been postulated no single one being adequate in explanation. It is probably true to state that in different cases one or more of the following factors may be responsible.

- (1) Growth disturbance
- (2) Infection
- (3) Embolic Vascular Occlusion
- (4) Trauma  $\left\{ \begin{array}{l} \text{Vascular Occlusion} \\ \text{Infarction} \end{array} \right.$

*Brailsford* reviews the various theories in great detail and believes that trauma is of paramount importance but admits that this alone will not account for the lesion in all cases—trauma alone obviously cannot be held responsible in cases which show multiple osteochondritic foci examples of which have been reported by *Martin* and *Roesler* and by *Pickett* and *Harbin* (It should be mentioned that when multiple lesions are noted in an individual case there is a tendency to classify such a case as a form of dyschondroplasia )

### MORBID ANATOMY

In a disease the course of which is limited at the cessation of growth biopsy is seldom justified—hence there has always been a paucity of material for pathological study

*Zemansky* in 1928 after an extensive study of the literature and of original material in osteochondritis of the femoral head described the usual changes thus (1) extensive subchondral necrosis of the bone and marrow (constant) (2) partial or complete destruction of the epiphyseal line (inconstant) (3) fragments of dead bone surrounded by richly vascularised connective tissue (4) fibrous tissue replacement of necrotic areas with osteoid formation from fibrous tissue and pre existing lamellæ *Zemansky* believes that the process follows a *traumatic vascular occlusion* of the femoral head epiphysis and suggests that these changes are those of infarction and its sequelæ

The fundamental change is that of aseptic necrosis of a growing epiphysis or short bone absorption of the necrotic tissue eventual replacement by normal bone Destruction of articular cartilage appears to be the exception rather than the rule but it seems reasonable to suggest that this will occur in untreated cases The greater the cartilage destruction the more marked will be the clinical signs and the greater the liability to resultant deformity and eventual osteo arthritis

One other description of the pathological process deserves serious consideration in juvenile osteochondritis although it will not be applicable in the adult bone lesions This description is that the primary change is one of growth disturbance with multiple infarctions in the affected area leading to epiphyseal collapse

### General Clinical Features

The clinical signs and symptoms vary considerably in different situations in different patients It is not an uncommon experience to find typical radiographic appearances of osteochondritis at a site presenting no clinical signs—further in certain cases with bilateral involvement of similar anatomical zones clinical manifestations may be unilateral

Whatever may be the role of trauma in the onset of the disease there is no doubt that once osteochondritis is established the degree of clinical severity will be greatly influenced by the stress and strain that may be borne by the



affected zone—e.g. involvement of the femoral head is likely to be clinically more significant than osteochondritis in the iliac crest epiphyses.

### *Radiological Observation in Osteochondritis*

Whenever possible, the course of an osteochondritic lesion should be followed by regular controlled radiographic studies. When a case is first seen, the technical factors employed should be noted, and these should be repeated at consequent examinations. If a case is followed over a long period, slight allowances may have to be made for increases in stature.

If an osteochondritic lesion is suspected, the same zone on the opposite side of the body should always be radiographed also, further, in certain cases it may be desirable to extend the investigation in order to recognise such conditions as that form of dyschondroplasia described by *Morquio* and *Brailford*.

## OSTEOCHONDRITIS IN DIFFERENT ZONES

The commonest sites in which osteochondritis occurs are the femoral head epiphyses, the vertebral epiphyseal plates, the tarsal scaphoid, the second metatarsal head, and the tibial tubercle. The two first-mentioned sites are probably those in which the disease has the greatest significance, and in which correct diagnosis in the early stages is of paramount importance in order to institute adequate early treatment. It is only by earlier and more prolonged treatment than is customary that the end results of osteochondritis of the spine and hip can be improved.

## OSTEOCHONDRITIS OF THE VERTEBRAL EPIPHYSEAL PLATES

*Synonyms*—*Scheuermann's disease*, juvenile kyphosis, kyphosis osteochondropathia, vertebral osteochondritis, vertebral epiphysitis, kyphosis dorsalis adolescentium, kyphosis dorsalis juvenilis (Osteochondritis may also affect the primary vertebral body ossific centres, a condition first described by *Calvé*—the resultant deformities in this form may be severe, it is a rare type, whereas the secondary centre type is relatively common).

*Clinical Features*—Certain cases of dorsal kyphosis in children are due to osteochondritis, the onset is usually at the age of puberty, and both sexes may be affected. The peak incidence age in *Scheuermann's* series of over 200 cases was from 15 to 16 years. Pain is usually present, and is associated with a characteristic sensation of "tiredness in the back." Long maintenance of the erect posture aggravates the pain, which is relieved on lying. The musculature of the back is not affected until an advanced stage has been reached.

*Normal Development*.—The plate epiphyses first ossify between the eighth and thirteenth years, the plates are best seen in the lower dorsal region, and are almost always demonstrable between 15 and 18 years. In lateral views, they are seen to be parallel to the upper and lower margins of the bodies;

anteriorly the plate is thicker and corresponds to the small anterior step in the body itself. The ossification should be smooth and regular.

**Radiographic Appearances** — *Scheuermann* has described the appearances in great detail. The zone of the tenth to eleventh dorsal vertebræ is that most commonly affected. There is seen a disordered ossification, so that the deepest



FIG. 402.—Osteochondritis of vertebral epiphyseal plates in the lower dorsal spine. Slight changes.

part of the plate is not apparent, the vertebral body becoming wedged, the thickest part of the plate may be displaced inwards or backwards and irregularities occur in the plate proper and in the apposing body surface. The body is plastic and develops further wedging (Fig. 402). The sequelæ in severer cases are those of hypertrophic spur development, and a typical spondylitis osteoarthritica may mask the initial disease process.

Typical Schmorl's nodes may be noted from prolapse and herniation of the nucleus pulposus of the disc. Certain cases of juvenile kyphosis may be due to this prolapse alone without an osteochondritis being present. There does not seem to be much justification for the controversy of *Scheuermann's* views versus *Schmorl's* views.

both are correct in different subjects and both play an important role in juvenile kyphoses.

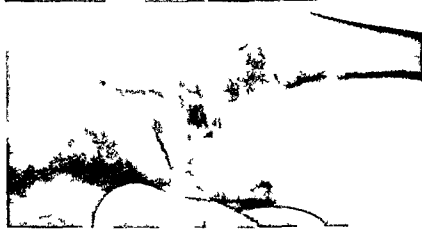
The radiographic appearance of established vertebral osteochondritis is so typical that there is no problem of differential diagnosis from other pathological processes. The difficulty lies in deciding whether the appearances in a suspected early case are within normal limits or whether definite pathology is present.



FIG. 403 (a).—Osteochondritis of left femoral head: boy aged 7 years: early stage, showing an increase in the density of the head with surrounding osteoporosis, but without flattening or fragmentation.



FIG. 403 (b).—Same case as (a) at 14 years. Good, but imperfect result: slight flattening present.



(a) 10404 Rn) gail e o l f o t e o d t of f e o a l l e a l l c o n t a t e a t n t (a) Jan ary 193 Slit  
 co l e n s e n o f l w t l n a l g u a r i t y (b) O t o b e r 1936 I r e g a r t y n o u t l n o a n d l o n t y (c) F e l a y 1937  
 L a y v i e a e y l a o p y s t l l a t

## OSTEOCHONDRITIS OF THE FEMORAL HEAD EPIPHYSIS

*Synonyms* — Perthes Legg's Calvé's Disease osteochondritis coxæ juvenilis osteochondritis deformans pseudo coxalgia

**Clinical Features** — Osteochondritis of the femoral head is much commoner in boys than in girls the age limits of the condition are 3 and 14 years the peak incidence age is from 6 to 8 years both femoral heads may be involved

The cardinal symptom in most cases is that of a lump pain is frequent but varies considerably in severity a history of trauma is frequently obtained On examination in the earliest stages it is found that the position assumed by the limb is that of slight adduction and flexion abduction and internal rotation may be limited slight muscular wasting may be present but true shortening is not detected

If the disease remains untreated the clinical course will depend according to Legg on the type of deformation that results

Legg recognises a mushroom deformity of the femoral head in which a lump is prominent but in which atrophy pain and shortening are slight and a cup deformity in which pain shortening and eventual limitation of movements are marked

There is no doubt that treatment in most cases is not so prolonged as it should be *Brailsford* has produced convincing evidence that although the



FIG. 40a.—Osteochondritis of femoral head following contusion and fixation of fracture of femur.

disease is self limiting during the recovery phase the epiphysis is "plastic" and is very susceptible to pressure trauma. This author demands that treatment should be maintained until epiphyseal regeneration is complete. This judgment can only be made by radiographic studies.

Most cases show a course in which the clinical and radiological findings may be tabulated (after *Brailsford*)

TABLE SHOWING CORRESPONDING CLINICAL AND RADIOLOGICAL PHASES IN OSTEOCHONDritis OF FEMORAL HEAD

Phase	Clinical Signs	Radiographic Signs
1	Marked	Minimal
2	Subside	Typical Fragmentation
3	Exacerbation	Deformation
4	Those of Arthritis in untreated cases	Those of Arthritis in untreated cases

**Radiographic Appearances**—These have been described by *Brailsford* in greater detail than by any other author after a controlled study of seventy one cases. The changes in chronological order are

(i) EARLIEST SIGNS (Figs 403a and 404a)

(a) Slight increase in density of femoral head



FIG 406—Advanced bilateral osteochondritis of femoral heads in an untreated case with epiphyseal widening

- (b) Slight surrounding osteoporosis
- (c) Slight increase in joint space
- (d) Upward convexity of the femoral neck (*Gage*)

(ii) ESTABLISHED DISEASE (Figs 404b 404c 405 and 406)

- (a) Fracturing fragmentation and compression of epiphysis with dense irregular bone islets
- (b) Gradual absorption of dense bone in epiphysis
- (c) Thickening of juxta epiphyseal diaphysis

(iii) RECOVERY STAGE (Figs 403b 404c)

- (a) Regenerating epiphyseal outline with continued disappearance of dense fragments
- (b) Osteoporosis in vicinity no longer apparent
- (c) Complete epiphyseal regeneration

The recovery stage lasts from two to four years and during the whole of this period the bone is in a plastic state and should not be submitted to full weight bearing

The final appearances of the end result will depend entirely on the degree of deformity suffered in the earlier stages. In untreated cases or in those in which treatment has been inadequate the head will be flattened, especially on its medial aspect and the margins will splay out and overhang the neck i.e. a typical mushroom appearance is seen. Hypertrophic arthritic changes will occur in young adult life in such cases.

**FURTHER OBSERVATIONS**—Acetabular osteochondritis is sometimes seen in association with typical femoral head osteochondritis. The acetabulum will show the density variations and slight compression but fragmentation is unusual.

Typical changes of osteochondritis in the epiphysis may sometimes be seen in reduced congenital luxations of the hip joint. *Hodges Phenister* and *Brunschwig* have seen cases in which adolescent cox vara is complicated by osteochondritis in the slipped head or in the metaphysis. Further they mention instances in which a slipped epiphysis was noted on one side and a typical head osteochondritis on the other side in the same patient.

**Differential Diagnosis**—In the fragmentation stage the appearance is so typical that difficulty in differentiation should seldom arise. In the stage of onset and recovery however the changes may superficially resemble those seen in

(i) **TUBERCULOUS ARTHRITIS**—Osteoporosis and cartilage destruction are usually marked. The periphery of the head will show a ragged erosion and sequestration is either very gross or microscopic so that it is not appreciable.

(ii) **HYPOTHYROIDISM** (cretinism and infantile myxœdema)—Coarse apparent fragmentation is seen actually the epiphysis forms from several

irregular ossific nuclei which are delayed flattening may be extreme, the changes are always bilateral and usually symmetrical

(iii) **POLYARTICULAR ARTHRITIS IN CHILDREN** (*Still's disease*)—An isolated hip joint radiogram may simulate osteochondritis. Epiphyseal irregularity is present but fragmentation does not occur, compression is not seen

(iv) **DYSCHONDROPLASIA** (*Morquio Brailsford form*)—In this condition, fragmentation may be present but severe metaphysical changes are also noted. The changes are symmetrical and are not confined to the hip joints

### OSTEOCHONDRITIS OF TARSAL SCAPHOID

(*Syn Kohler's disease*)

**Clinical Features**—This condition is not uncommonly found in children between the ages of 3 and 10 years, there is a decided peak incidence at 5 to 6



FIG. 407—Osteochondritis of the tarsal scaphoid shortly after appearance of ossific nucleus. Note general tarsal osteoporosis.

years. Boys are more commonly affected than girls. A definite history of trauma is only obtained in a minority of cases. The clinical signs are usually those of a low grade inflammatory lesion, however, the radiographic discovery of the lesion may be accidental, the condition may affect both feet.

**Radiographic Appearances**—The early appearances of X-ray examination depend upon the degree of ossific growth of the nucleus when the disease begins. In the younger patients the small ossific nucleus undergoes a coarse fragmenta-



tion and thus together with neighbouring osteoporosis is the earliest sign (Fig 407) In older cases, when the scaphoid is already well-developed the first change is one of simple increase in density without alteration in shape (Fig 408) Antero posterior compression and fragmentation then occur, typically the scaphoid appears as a disc with irregular margins and with a dense central fragmented core

In the recovery phase the central density disappears and an envelope of new bone is formed (Fig 409) The recovery of form may be perfect but



FIG 408—Osteochondritis of tarsal scaphoid early stage showing an increase of density without deformity

dorsally irregular extrusions may be seen (Fig 410) A striking feature is that the normal gap between the astragalus and first metatarsal base is seldom shortened thus at least suggests that the surrounding cartilage is not markedly necrosed The recovery is usually complete in a period of eighteen to twenty four months

### OSTEOCHONDRITIS OF METATARSAL HEADS

(Syns Kohler's disease Freiberg's infraction)

**Clinical Features**—The second metatarsal head epiphysis is much more often affected than the heads of other metatarsals The condition is much

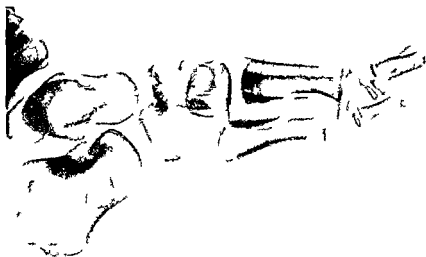


FIG 409 Osteochondritis of tarsal scaphoid showing degeneration about a fragmented core



FIG 410 Osteolysis of tarsal apophysis. Note the lateral fragment extrusion

commoner in girls than in boys. The usual age incidence is from 10 to 15 years but cases have been reported in which the onset occurred in adult life. Trauma is usual in the history; the signs are those of a low grade inflammatory lesion. Osteochondritis in this site has been studied more often by biopsy than in any other area.

**Radiographic Appearances** — The primary changes in the metatarsal head are those of osteochondritis in other bones, i.e. condensation, fragmentation and deformation. There is usually an increase in the joint space. Secondary changes are especially pronounced at this site and are characteristic: the base of the proximal phalanx hollows out centrally and splays out at the margin as if gross osteoarthritis; the metatarsal shaft shows a marked thickening distally as a result of regular deposition of periosteal new bone. In this way the tubular part of the second metatarsal takes on the characters of the first metatarsal.

One feature is commonly seen at this site which is unusual in osteochondritis elsewhere, namely the head fragments which may be large, may separate completely and avoid inclusion in the recovery phase and hence remain as loose bodies in the joint (Fig. 411).

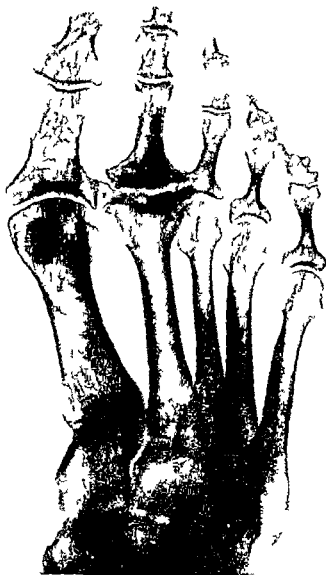


FIG. 411.—End result of osteochondritis of the 1st metatarsal head. Note the large loose body in the joint and the normal mass of the shaft.

## OSTEOCHONDRITIS OF THE TIBIAL TUBERCLE

(Syns *Schlatter's disease* *Osgood's disease*)

**Clinical Features**—This condition is commoner in boys than in girls the usual age incidence is from 12 to 15 years Trauma is nearly always a distinct

feature in the history It is indeed difficult to decide in certain cases whether an abnormality of the tibial tubercle should be classified as a true separation fracture or as an osteochondritis

**Anatomical Development**

—This should be fully understood in its different forms The proximal tibial epiphysis ossifies at or just before birth In infancy the tubercle zone is represented by a small tongue of cartilage which continues to grow downwards Secondary ossification does not occur in this lingula until puberty it is very variable in form a separate centre or multiple centres may form the whole lingula—more commonly the ossification is an extension from the main proximal epiphysis and this extension may be complete or partial, if the latter then a separate nucleus is usually present for the interior extremity of the lingula (*Fels*)

**Radiographic Appearances**—From the description of the anatomy given

above it will be realised that osteochondritis will not be easy to diagnose with confidence the sign of fragmentation will obviously be difficult to assess—density variations in the various nuclei (i.e. multiple) together with an uneven



FIG. 413.—Osteochondritis of the tibial tubercle—regeneration phase

elevation (forward protrusion) of them will prove the more reliable guides (Fig 412)

In this as in other osteochondritic processes it is important to make serial studies. When the condition is bilateral the writer has noted that the side showing the greater radiographic signs does not necessarily manifest more marked clinical signs.

Detailed and full studies of the condition have been made by *Sellatter*, *O'good* and *Fels*.

### OSTEOCHONDRITIS IN OTHER SITES

*Osteochondritis of the Calcaneal Epiphysis* is sometimes a cause of painful heel but is not as common a condition as certain reports would lead one to expect. Normal ossification may be markedly multinucleate and appear to be irregular in consequence the association of anatomical irregularity with painful heel does not justify a diagnosis of osteochondritis without careful serial examinations.

*Osteochondritis of the Patella* may affect the primary centre in young children (*Köhler*) and also the secondary epiphyseal centres in children from the age of 10 to 14 years (*Sinding-Larsen*, *Johansson*).

*Osteochondritis of the Neck of the Femur* is an interesting condition in view of the part it may play in the development of the so-called *infantile coxa vara*. Certain anatomists believe that secondary ossification centres occur in the femoral neck cartilage but there is no unanimity of



FIG 413.—Infantile coxa vara post-traumatic osteochondritis of the femoral neck.

opinion on this point *Gutig* and *Hertzog* and *Brailsford* have reported cases in which infantile coxa vara resulted from a process resembling osteochondritis both clinically and radiographically which did not affect the head epiphysis (Fig. 413)

### OSTEOCHONDRITIS DISSECANS

*König* in 1887 was the first writer to describe a peculiar form of loose body formation in joints which he named osteochondritis dissecans. In this condition a necrotic process separates a bone fragment lying beneath the articular cartilage which is eventually also involved the osseocartilaginous fragment may then separate completely and form a loose body. The aetiology is not definitely understood but trauma is undoubtedly responsible in certain cases. The condition may be recurrent in the same joint.

**Clinical Features**—Osteochondritis dissecans is much commoner in males than in females and is usually seen in young adults. The lesion is more commonly noted in the knees than in other joints but it has been reported showing involvement of the elbow (Fig. 414) and shoulder joints. When the knee is affected the condition may be bilateral however only one side may give clinical signs.

The clinical history is usually one of old trauma with consequent recurrent attacks of internal derangement of the knee joint i.e. synovitis with effusion locking and local pain. The certain diagnosis of osteochondritis dissecans can only be made radiographically other than by operation.

**Radiographic Appearances**—In the early stages before the separation of bone from bone is complete the condition may be very difficult to define unless several views of the joint are taken in different

degrees of rotation as well as in the standard positions. Once separation of bone from bone is complete the appearances become typical. A rounded or oval bone fragment the margins of which are not sharply defined is noted if seen *en face* a translucent ring will be seen surrounding the bone fragment



FIG. 414 Osteochondritis dissecans of the capitulum

if seen *en profile*, the fragment will be noted lying opposite a depression in the femoral condyle. The medial femoral condyle is affected more frequently than the lateral (Fig. 415).

In the later stages, if the condition progresses, cartilaginous continuity will be interrupted and the complete osteocartilaginous fragment will be noted as



FIG. 415.—Osteochondritis dissecans of the medial femoral condyle. O—ous continuity broken but fragment not separated.

a loose body in the joint. the site of origin will be seen, for a limited period, as a depression.

If the lesion fails to develop, then a gradual return to normal may follow, i.e. the translucent zone gradually becomes obliterated by new bone formation and no separate fragment can be identified.

#### BIBLIOGRAPHY

- ANDERSEN, G., *Arch. J. Clin. Chir.*, 1924, CXXIX, 26.  
 BRANFORD, J. F., *Brit. Journ. Radiol.*, 1935, VIII, 87.  
 BUCHMAN, J., *Journ. Bone and Joint Surg.*, 1925, VII, 814.

- BURNS B H, *Proc Roy Soc Med*, 1931, XLIV, n, 912  
 BUSCHKE F, *Acta Padiol*, 1934, XI, 502  
 CALVE, J., (1) *Jour de Padiol et D Elect*, 1925, 9, 22, (2) *Peu de chir* 1910 XLII 24  
 FELS E, *Arch f klin Chir*, 1924, CXXIX, 552  
 FREIBERG, A H, *Surg Gynec and Obstet* 1930 LI, 145  
 FRIEDRICH H, *Deutsch Ztschr f Chir*, 1924, CLXXXII, 385  
 GAGE, H C, *Brit Journ Padiol*, 1933, VI, 295  
 GUTIG C, and HERTZOG, A, *R ntgenpraxis*, 1932, IV, 504  
 HARBIN, M, and ZOLLINGER, R, *Surg Gynec and Obstet*, 1930, LI, 145  
 HODGES P C, PHEMISTEP, D B, and BRUNSCHWIG, A, *Diagnostic Roentgenology*, 447  
 Nelson New York 1936  
 JOHANSSON, S, *Ztschr f Ortho Chir*, 1924, XLIII, 82  
 KIENBOCK R, *Fortschr a d Geb d R ntgenstr*, 1910, XVI, 77  
 KOHLER A, *Munchen med Wchnschr*, 1903, LV, 1923  
 KONIG, F, *Deutsch Ztschr f Chir*, 1887, XXVII, 90  
 LEGG A T, *Boston Med and Surg Journ*, 1910 CLXII, 202  
 LEWIN P, *Journ Bone and Joint Surg*, 1927, IX, 456  
 MARTIN W C and ROESLEE, H, *Amer Journ Roentgen*, 1931, XXVI, 861  
 MAUCLAIRE, M, *Bull et Mem Soc Nat de Chir*, 1927, LIII, 1377  
 MONDE F, *Beitr z klin Chir*, 1922, CXXVI, 707  
 MOUCHET A, *Bull et Mem Soc Nat de Chir*, 1928, LIV, 986  
 OSGOOD R B, *Boston Med and Surg Journ*, 1903, CXLVIII, 114  
 PERTHES G, (1) *Deutsch Ztschr f Chir*, 1910, CVII, 111, (2) *Arch f klin Chir* 1913  
 CI 779  
 PICKETT J C, and HARBIN, M, *Surg Gynec and Obstet*, 1933, LXI 1000  
 PREISER G, *Fortschr a d Geb d R ntgenstr*, 1910, XVI, 360  
 RENANDEP, A, *Acta Padiol*, 1924, III, 521  
 RITTER, quoted by LEWIN, *Amer Journ Dis Children* 1929, XXXVII, 141  
 SCHPUERMAN, H, (1) *Ztschr f Ortho Chir*, 1921, XLI, 300, (2) *Fortschr a d Geb d  
 R ntgenstr* 1936 LIII, 1  
 SEVER J W, *New York Med Journ*, 1912 XCV, 1025  
 SINDING LARSEN, C F, *Acta Padiol*, 1921, I, 171  
 VAN NECK, M, *Arch Franco Belg de Chir*, 1924, XXVII, 238  
 VOLTANCOLI, G, *Chir d Org di Mori*, 1925 IX, 281  
 WAGNER A, *Acta Padiol*, 1930, XI, 80  
 ZEMANSKY, A P, *Amer Journ Surg*, 1928, IV, 169



## PART THREE

### SECTION VII

#### STATIC AND PARALYTIC LESIONS THE INTERVERTEBRAL DISCS ORTHOPÆDIC OPERATIONS

BY

F CAMPBELL GOLDING, M.B., M.R.C.P., D.M.R.E.

#### CHAPTER XXXIV

##### STATIC AND PARALYTIC LESIONS OF BONES AND JOINTS

##### STATIC DEFORMITIES

THE RESULTS produced by postural defects depend to some extent on the age of the patient at the onset. The young patient may develop a severe deformity.

A postural defect leads to an anatomical fault and results in a limited bony surface bearing excess of weight. The relative position of the bones is altered producing structural changes which predispose to secondary deformities of an arthritic type.

In all these deformities the same principle may be applied, that the part bearing excessive pressure suffers some retardation of development. The internal architecture of the bone may also be altered to compensate for the alteration in the distribution of body weight.

**Scoliosis**—This is a condition of lateral curvature of the spine. It may be due to a known cause, e.g. congenital deformity of the spine, empyema or fibrosis of lung, torticollis or any condition causing shortening of one leg or tilting of the pelvis. A larger group is formed by cases of idiopathic or static scoliosis, the cause of which is frequently unknown. The onset of the condition is usually in adolescence.

In the early stages the scoliosis disappears on lying or may be corrected by muscular effort, and no radiographic changes are visible. Later the deformity becomes irreducible. At this stage the condition usually consists of a dorso-lumbar curvature, with the convexity directed to the right. There may be secondary curvatures above and below the deformity, in the opposite direction (Fig. 416). The bodies are rotated towards the convexity and the spinous processes incline towards the concavity. The appearance of the vertebrae is normal, apart from rotation. At a later stage the bodies become wedged and

the intervertebral space may be narrowed on the concave side. Associated with the scoliosis are secondary changes in the shape of the ribs and pelvis.

The deformity in the spine predisposes to the early development of osteoarthritic changes (Fig 417). The osteophytic deposits are usually more prominent on the concavity of the curve.



FIG 416 A Curve which the surgeon is attempting to limit by a spinal graft.



FIG 417 Lumbar scoliosis with rotation of the body and osteoarthritic change.

**Genu Valgum Genu Varum**—The radiological appearances in these conditions are frequently less striking than the clinical examination would suggest.

There may be slight narrowing of the outer or inner half of the epiphyses with corresponding overgrowth of the inner or outer border of the metaphysis of the femur and tibia. The changes are more marked in the femur than in the tibia and the metaphyseal overgrowth accounts for the major part of the deformity.

**Flat Foot**—In flat foot there is some abduction and pronation of the foot at the mid tarsal joint. The radiological changes are seen best in the lateral view (Fig. 418). The most obvious of these are

(1) The scaphoid and cuboid slip downwards. The scaphoid may be wedged and the upper part of the joint space between it and its neighbours may be widened.

(2) The astragalus appears foreshortened with a prominent anterior and upper margin. Its anterior extremity is depressed.



FIG. 418.—The deformities resulting from a flat foot which developed at an early age.

(3) In advanced cases a line drawn from the postero-inferior angle of the calcaneus through the lower margins of the cuboid tuberosity and head of the fifth metatarsal is horizontal or even convex downwards.

## PARALYTIC DEFORMITIES

The skeletal manifestations of paralytic lesions depend upon the site of the lesion and to some extent on the function of the area affected.

**Lesions of the Upper Motor Neurone**—The bones show the usual changes associated with disuse. During the growing period the shape of individual bones e.g. in the foot may be altered by a spastic paralysis in much the same way as described in static lesions. Individual metatarsals may hypertrophy in a remarkable fashion when they are forced to bear unusual strain.

A severe spastic paralysis may cause subluxation of a joint. Spastic dyslegia is due to agenesis or arrested development of the cells of the pre-Pyramidal area. The spasticity of the lower limbs may cause a subluxation at the hip joint—a change which is visible on the radiogram. Severe scoliosis may be caused by both lower and upper motor neurone lesions e.g. poliomyelitis, Friedrich's ataxia and syringomyelia.

**Lesions of the Lower Motor Neurone and of the Nerves**—The changes introduced in bones and joints may be the result of the flaccid palsy, the defective proprioceptive sense or the loss of the trophic influence of the nerve. In some cases there is the combined effect of more than one of these factors. One of the simplest types of joint deformity is the dislocation produced by muscle



FIG 419 The skeletal deformity due to anterior poliomyelitis

palsy for example at the shoulder by paralysis of the shoulder girdle muscles

A joint may be deformed by the constant trauma associated with subluxation. It is possible to produce a subluxation or dislocation at the hip joint in some patients who have made a partial recovery from poliomyelitis. This frequently results in a deformed acetabulum which simulates the appearance of congenital dislocation.

More extensive joint changes are found in a group of progressive

FIG 410 —Leprosy. There is partial absorption of the terminal phalanx of the 1st toe with an opaque salt lying in a ulcer. The terminal phalanx of the 2nd toe is missing. The 1st and 2nd metatarsal phalangeal joints show neurotrophic change. There has been a fracture of the 1st metatarsal.

diseases of the nervous system and are referred to as Charcot's joints (see Chapter XXX pp 446-9).

The changes in bones are of several types. Examples of arrested development are seen in cases of anterior poliomyelitis. This disease results in necrosis of grey matter in the spinal cord, brain stem, cerebrum and cerebellum with practically no sensory disturbance. In severe cases the bones are defective in length and width. The radiological findings are often particularly marked in the upper limb. The bones are sometimes much thinner than normal



FIG 411 Multiple deformities in syphilis with perforating ulcers

with a relative increase in width of the cortex. The cancellous structure is thin and coarse in the region of the expansion at the joints. The bones are liable to fracture. Examination of the soft tissues shows narrow strips of muscular tissue with a disproportionate increase in subcutaneous fat (Fig. 419).

In certain conditions such as tabes syringomyelia and leprosy the bones exhibit abnormal fragility and spontaneous fractures may occur. These may often be found accidentally in the absence of a history of fracture. They are not necessarily confined to the region of a joint (see Charcot's joint). Union does not always occur and a pseudarthrosis may form. The structure of the bone appears normal or there may be slight osteoporosis. There is no demonstrable local cause which determines the site of fracture.

Marked osteolytic changes are often seen in the small bones of the hands and feet. The most typical appearance is a short and pointed phalanx from a partial absorption which has commenced at the distal end of the bone. As bone is destroyed it is absorbed (Fig. 420). The condition may be complicated by secondary infection from a perforating ulcer usually under a metatarsal head. In these patients some sclerosis and periosteal new bone formation may be added to the absorption and the metatarsophalangeal joints may be completely disorganised (Fig. 421).

## CHAPTER XXXV

### THE INTERVERTEBRAL DISCS

THERE IS a progressive change in the radiographic appearances of the normal intervertebral disc from infancy to old age. In the six months foetus the cartilage space is almost as wide as the body. In the adult the discs form approximately one quarter of the spinal column, they are widest in the lumbar region and narrowest in the upper thoracic spine. In old age the space is further reduced by relative desiccation and degeneration of the matrix.

#### The Normal Disc

Some knowledge of the anatomy is necessary in order to understand the changes produced in the discs and vertebrae by trauma, degeneration and disease (see also Vol. III Part III Section I). The intervertebral discs are composed of three parts—the disc plates, the annulus fibrosus and the nucleus pulposus.

*The disc plates* are two thin layers of hyaline cartilage which lie over the central perforated area of each vertebra. They enclose the nucleus pulposus. At the periphery they are bounded by the raised margin of the bodies which is formed by the united epiphysis. This epiphysis is an incomplete bony ring lying on the antero-lateral margins of the bodies. Its time of appearance and union with the body vary with the individual and with the sex, being earlier in girls than in boys. It is frequently stated that it appears in adolescence but radiograms show that many patients develop bony nuclei at eight years or even earlier. Union takes place usually before the age of 20 but is very variable. On the lateral radiogram it shows as a small triangular fragment of bone at the upper and lower angles of the anterior surface of the bodies. These epiphyses take no part in growth but serve to unite the fibres of the annulus fibrosus to the body.

*The annulus fibrosus* consists of lamellated fibrocartilage bounded by fibrous tissue. The outer rings are firmly attached to the margins of the vertebrae. The inner fibres blend with the substance of the nucleus pulposus. The annulus serves as a limiting capsule of considerable strength.

*The nucleus pulposus* is an incompressible oval ball of high fluid content and gelatinous consistence. It contains loose bundles of fibrous tissue with mesenchymal cells. The demarcation between the nucleus and the annulus becomes less distinct with advancing years.

## The Abnormal Disc

Deformities of the disc are produced by

### I Congenital abnormalities

- (a) Absence of the disc
- (b) Congenital deformities in the bodies
  - (i) Local deformities
  - (ii) Dystrophies

II Degenerative and traumatic lesions The relative importance of these two factors is often difficult to determine—either or both may be present as the primary factor, or may be superimposed on a congenital abnormality

- (a) Nuclear expansions of adolescence
- (b) Senile degeneration
- (c) Calcification
- (d) Trauma from lumbar puncture
- (e) Herniation into the body
- (f) Herniation into the vertebral canal

### III Changes produced by disease

- (a) Infective diseases
- (b) Rheumatic conditions
- (c) Malacic processes in the vertebrae
- (d) Osteochondritis

## CONGENITAL ABNORMALITIES

*Absence of the disc* may be partial or complete. The incomplete disc is usually due to fusion of the anterior margins of the bodies (Fig. 422). Complete fusion is sometimes called "block vertebra". Any part of the spine may be affected. Sometimes the spinous processes are fused.

The diagnosis has to be made from an infective lesion which has caused ankylosis. This may be assisted by the absence of a suggestive history, or by the absence of structural change in the bodies.

A *primary congenital defect in the spine* leads to irregularities in the disc space. In this case the abnormality of the disc is obviously of secondary importance to the presence of hemivertebrae or bipartite bodies, etc.



FIG. 422—Partial fusion of two vertebrae due to a congenital anomaly.



FIG 4-3 Deformity of the bodies in chondroosteodystrophy

in old age. These patients exhibit a progressive fibrillar degeneration with some necrosis at the points of pressure. It may be seen in a pure form in senile kyphosis. The disc becomes narrow towards the anterior border and in severe cases the vertebrae may be almost in contact.

**Calcification**—Large or small oval opacities are occasionally found in the region of the nucleus. The whole nucleus may be calcified. The condition is seen best in the lateral film (Fig 4-4).

Small opacities may form and ossify in the annulus at the periphery of the disc space usually anteriorly. From the film alone it is difficult to determine the proportion deposited in the annulus or ligaments. Probably the major part is in the latter. Opacities in this region may be due to deposits of calcium which are structureless or they may be composed of bone when they are sometimes referred to as *intercalary bones*.

**Lumbar Puncture**—Several cases have been reported recently by Milward and Grout and by

*Chondro osteo dystrophy* is a congenital hereditary and familial disease. Epiphyses are particularly affected and the vertebral epiphyses do not escape.

In childhood the discs are frequently thicker than normal and the outlines of the vertebrae are deformed. The anterior part of the body is frequently beveled (Fig 4-3). In later life the disc space is narrower than normal with a marked irregularity of the margins of the vertebrae associated with changes in the disc plate and subchondral bone.

#### DEGENERATIVE AND TRAUMATIC LESIONS

**Nuclear Expansion** The cause of nuclear expansions in adolescence is not known. The diameter of the expanded area is seldom more than 1 cm. The condition does not appear to be of any importance.

**Senile Degeneration**—It has been shown by Beadle, Ross, Smith and others that the discs are particularly prone to degenerative change.



FIG 4-4—Partial calcification of three nuclei



*Pease* in which changes in discs occurred after lumbar puncture presumably from actual puncture of the disc. The disc space is thin and sometimes irregular. Osteoarthritic changes may appear rapidly. It is difficult to understand the marked irregularity of the subchondral bone in these cases. The narrowing of the disc with subsequent osteoarthritis is compatible with escape of the nucleus through a perforation of the annulus by the needle. The irregularity of the vertebral surface due to subchondral changes is possibly due to introduction of sepsis but several patients who exhibited these changes after lumbar puncture for spinal anaesthesia did not show other evidence of infection.

*Herniation into the Body Schmorl's Node*—Since the researches of *Schmorl* this term has been frequently used for herniations of part of the nucleus through the cartilaginous plate into the vertebral body. *Schmorl* found the condition in 38 per



FIG 43.—*Schmorl's* nodes in several vertebrae. Note the compact bone at the periphery of each node.



FIG 46.—A well defined herniation of the nucleus pulposus into a vertebral fracture.

cent of post mortem specimens. The predisposing factor appears to be a defect in the cartilaginous plate (which may be developmental or degenerative in origin) associated with the slight recurring trauma to which the spine is subject. The nodes vary in size and appear as punched out areas which are frequently symmetrical in neighbouring bodies (Fig 42c). In the recent state most are overlooked as the margins are not defined and normal cancellous bone is superimposed over the area. In the later stages they are outlined by compact bone which forms around the periphery or some calcification may be visible in the node itself. Diminution in the width of the

disc depends upon the amount of tissue lost. Most herniations do not cause any appreciable narrowing of the space but they predispose to further degeneration.

It is fairly characteristic of vertebral fractures that the disc space is preserved although its outline is deformed. In severe fractures some of the nucleus may escape into the body with narrowing of the disc (Fig 496). In less severe injuries herniations may be detected some months after an injury to the spine whereas immediate radiograms may not reveal these nodes (*Schmorl Joplin*). See Figs 302 303 Chapter XXV.

*Herniation into the Vertebral Canal*—For many years posterior herniations of the nucleus have been confused with benign tumours (chondromata) growing from the disc. Both conditions may occur but herniation is more common. The node projects to one side of the posterior longitudinal ligament in front of the dura.

The cause is unknown but it is possibly due to recurrent trauma in the presence of some defect in the annulus and is also influenced by the fact that the annulus is thin posteriorly. Herniations may occur anywhere but are most common in the lower lumbar spine. Multiple protrusions have been recorded (*Lore and Camp*). The patient may complain of sensory symptoms such as root pain, pain in the back, lumbago or intractable sciatica and in a late stage motor symptoms with disturbance of sphincters.

The direct radiological diagnosis is not possible in the majority of patients. Narrowing of the disc space is not constant and calcification in the node is uncommon. The diagnosis rests on a raised protein content in the cerebrospinal fluid (although this is not always found) and on the presence of a constant indentation of the thecal shadow in films taken after the injection of lipiodol (J c c) into the theca.

#### CHANGES PRODUCED BY DISEASE

Many of the changes due to disease affecting the disc are the secondary effect of vertebral involvement and are described in the appropriate chapters. The appearance of the disc depends upon the behaviour of the cartilaginous plates. The resistance of this layer of cartilage is considerable and some conditions such as secondary deposits may cause collapse of the body without invading the disc space.

*Most infective lesions* ultimately break through the plate and cause narrowing of the disc. Primary infection of the disc is externally rare as vascular channels are scanty and disappear when growth ceases but revascularisation may occur through a break in the plate or through a cartilaginous node projecting into the body.

*Rheumatic Diseases*—There is a type of lesion commonly found in patients who attend Rheumatic Centres which is believed to be due to the spread of infection from foci such as infected teeth or sinuses etc. These non specific

infective lesions are characterised by involvement of a single joint, with loss of cartilage and local sclerosis of bone. Changes of this type are occasionally found in the spine. The peculiar feature of this condition is a unilateral loss of the disc space, with subsequent sclerosis of the margins of the vertebrae and new bone formation. In the final stage the lesion resembles osteo arthritis, but it is localised to one disc, and the onset is much earlier than the customary age for this disease.

In spondylitis ankylopoietica, the calcification of the intervertebral ligaments and peripheral layers of the annulus may be clearly shown in a film. Ultimately the disc becomes ossified—it is then able to resist strain and retains its normal width.

*Malacia of the Bodies*—Any process causing osteoporosis or softening of the body may be accompanied by marked changes in the disc. In practice this is found most frequently in senility with osteoporosis (Fig 427). The natural turgor of the disc, acting on a softened spongiosa, causes a pressure deformity of the bodies, which are flattened and vary in depth or the discs become biconvex and the vertebrae resemble those of the fish. It is frequently difficult to obtain satisfactory radiograms of these patients, as the calcium content of the bodies is so low. A crush fracture is often superimposed on this condition.



FIG 427—A comparatively early stage in osteoporotic collapse of the bodies. The discs are assuming a lenticular form.

#### REFERENCES

- BEADLE O A, *Medical Research Council Special Report, Series No 161*, London, 1931.  
 JOPLIN R J, *Surg Gynec and Obst*, 1935, LXI, 591.  
 LOVE J, GRAFTON, and CAMP J D, *Journ Bone Joint Surg*, 1937, XIX, 776.  
 MILWARD F J and GROUT, J L A, *Lancet*, 1936, CCXXI, 183.  
 PEASE C N, *Amer Dis Child* 1935, XLIX, 840.  
 ROSS SMITH, N, *Brit Journ Surg*, 1931, XVIII, 358.  
 SCHMORL, G, *Fortschr Geb d R nt*, 1928, XXXIII, 265.

## CHAPTER XXXVI

### ORTHOPÆDIC OPERATIONS

THE PROCEDURES employed in orthopædic surgery vary in individual clinics and are constantly being modified as new or improved methods are evolved.

In the following brief description, those measures are considered which may be of radiological importance.

Confusion may arise in some instances between the disease process and the result of surgical intervention. The radiologist may also wish to follow the rationale of various operations.

#### OPERATIONS ON THE HIP

**Osteotomy.**—The object of the operation is to correct an angular or rotational deformity. It may be one of the following types (Fig. 428).

**SLITROCHANTERIC OSTEOTOMY**—A simple transverse section is made below the lesser trochanter. It is frequently performed for the flexion-adduction deformity of late infective arthritis.

**INTERTROCHANTERIC OSTEOTOMY**—The line of section is above the lesser trochanter where more extensive bone apposition may be secured. There are two types of intertrochanteric osteotomy.

(i) *Edge Osteotomy*—A wedge is removed from the outer side. The limb is abducted and the coxa vara or the adduction deformity is corrected.

(ii) *McMurray's Osteotomy*—The line of section is inwards and slightly upwards just below the neck. The object is to displace the whole shaft medially under the neck. This alters the line of weight bearing and permits the correction of deformity in osteoarthritis, it also promotes union in fractures of the neck (Fig. 429).

**THE LORENZ BIFURCATION OSTEOTOMY**—The operation is performed in upward dislocations with gross displacement. The object of the operation is to provide stability with a useful range of movement and to correct flexion and adduction. It increases the true shortening already present.

The line of section is inwards and sharply upwards at the level of the lower border of the empty acetabulum, into which the pointed lower fragment is thrust. Union is secured in abduction (Fig. 430).

**Arthrodesis**—The object is to produce a painless bony ankylosis.

**INTRACAPITULAR ARTHRODESIS**—The opposing joint surfaces are removed. The operation may be supplemented by inserting a Smith-Petersen pin through the femoral neck into the ilium (Watson Jones) (Fig. 431).

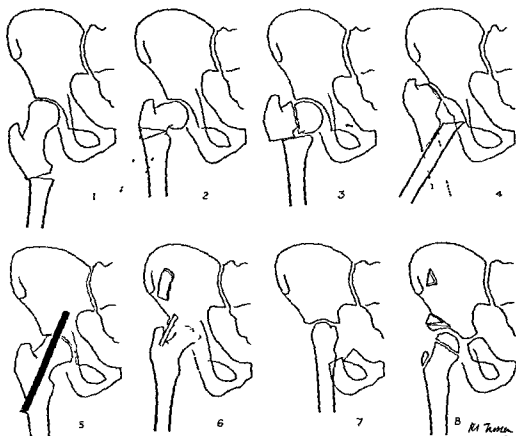


FIG. 428

- 1 Subtrochanteric osteotomy
- 2 Intertrochanteric wedge osteotomy
- 3 McMurray's osteotomy for fractured neck of femur
- 4 Lorenz bifurcation osteotomy for congenital dislocation.
- 5 Intra-articular arthrodesis with insertion of a Smith-Petersen pin
- 6 Extra-articular arthrodesis
- 7 Bankart's excision of the hip
- 8 Shelf operation

**EXTRA-ARTICULAR ARTHRODESIS**—Arthrodesis is obtained by a bony bridge from the femur to the ilium, above the joint. The reversed greater trochanter (Fig 432) and bone from the tibia have been used as grafts. The most satisfactory bridge is obtained by removing a complete rectangular island from the blade of the ilium, and wedging this into prepared clefts in the greater trochanter and the ilium above the acetabulum (Seddon) (Fig 433).

**COMBINED INTRA- AND EXTRA-ARTICULAR ARTHRODESIS** consists of an excision of joint surfaces reinforced by a bone graft.



FIG 429 —McMurray's osteotomy (for old Perthes disease)



FIG 430 —Lorenz bifurcation osteotomy



FIG 431 —Intra-articular arthrodesis supplemented by a pin (Watson Jones)



FIG 432 —Arthrodesis using the reversed trochanter (Hibbs)



FIG. 433 —Extra articular arthrodesis, using iliac graft (Nelson)



FIG. 434 —Shelf operation over a false acetabulum

**Excision of the Hip Joint** —The object of this operation is to remove all diseased tissue. It is usually performed in chronic tuberculous disease.

**SIMPLE EXCISION** —The remains of the head and neck are removed down to unaffected bone. The diseased acetabulum is curetted. A useful range of movement is frequently obtained with some shortening.

**RADICAL EXCISION (BANKART)** —The object of the operation is to shorten the course of the disease in chronic cases of tuberculosis of the hip joint with much destruction and to obtain some mobility.

The ilium and rami of pubis and ischium are divided beyond the diseased area. The whole acetabulum is removed. A variable amount of the femur is excised according to the extent of the process. Early movement is encouraged. A new joint forms. The patient may retain 90 degrees of flexion and a useful range of other movements.

**Arthroplasty** —The object is to restore movement. The operation is most frequently done on one side for patients with bilateral ankylosis resulting from septic arthritis or spondylitis ankylopoietica. The head and neck are refashioned by free removal of bone. Early movement is essential.

**Shelf Operation (Fairbank)** —After reduction of a congenital dislocation the defective upper rim of the acetabulum may not re-form especially after the age of 6 years.

The object of the operation is to ensure stability by deepening the acetabulum.

bulum with a buttress superiorly. When reduction is impossible, the operation may be performed over the false acetabulum (Fig 434). An iliac graft is wedged into a curved cleft over the acetabular margin.

**The Insertion of a Smith-Petersen Pin**—The common indication for the operation is the subcapital fracture. The radiologist is expected to provide films of reasonable quality in two planes with accelerated dark room technique. A Lasholm grid and small cone are essential accessories.

The results of the operation provide many interesting radiological appearances.

The points which should be recorded are:

*Before Operation*—(i) Rotation of the head usually an inward rotation. Complete reversal of the head may occur. (ii) The degree of varus. (iii) The amount of external rotation of the limb as shown by the position of the lesser trochanter. (iv) In late cases, any absorption of the neck or sclerosis of the head from defective vascular supply.

*During Operation*—(i) The extent of the reduction of the fracture, (ii) the position of guide wires, (iii) the position of the pin in relation to the neck and to the head and (iv) the final position of the fragments—especially the presence of impaction.

*After Operation*—Sequelæ which may be seen are: Non union, aseptic necrosis of bone round the pin usually at its extremities, erosion of the pin, fracture of the pin, loosening and expulsion of the pin, aseptic necrosis of the head, traumatic arthritis.

## OPERATIONS ON THE KNEE

**Supracondylar Osteotomy**—The object is to correct deformity, the commonest being genu valgum. The femur is divided transversely half an inch above the adductor tubercle through a short incision on the inner side of the limb.

To appreciate the final position films including half the femur and tibia are necessary. Non union is almost unknown.

**Arthrodesis**—This operation is seldom performed before the age of 16. A deformity may occur from disturbance of the epiphyseal lines. In the adult transverse resection of the joint surfaces is carried out. The patella may be used as an anterior bony reinforcement.

**Excision of Patella**—The patella may be excised in the rheumatic arthritides, fractures or in the operation of arthrodesis.

## OPERATIONS ON THE TARSUS

**Arthrodesis**—The objects of arthrodesis are usually to correct deformity to increase mechanical stability, and to relieve pain.



Common indications are neglected congenital club feet, paralytic conditions, and arthritis following fracture of the os calcis. The operation is frequently supplemented by tendon transplantation to restore muscle balance.

The tarsal regions of surgical importance are the subastragaloid, the astragalo-scapoid, and the calcaneo-cuboid joints. These act as one joint complex. Excision of any one single joint is considered unsound. Triple arthrodesis is commonly performed. It consists of excision of cartilage and a layer of bone from all the above joint surfaces. By excising the joints more freely on one side or the other, various deformities may be corrected. Fixation in plaster for at least three months is necessary to ensure bony fusion (Fig. 435).

Modifications of triple arthrodesis are

(a) NAUGHTON DUNN'S OPERATION.—In this the scaphoid is excised and

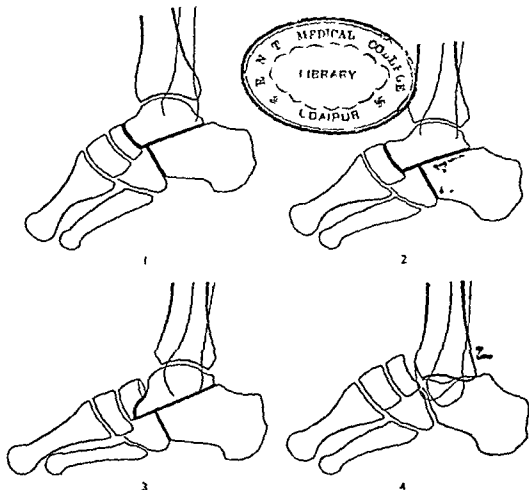


FIG. 435.—Diagrams of operations on the ankle joint.

1 Triple arthrodesis

2 Naughton Dunn's operation

3 Lamborn's operation

4 Whitman's astragalectomy

union secured between the astragalus and cuneiforms. This allows the foot to be displaced backwards in relation to the astragalus, and lessens the tendency



FIG. 436 —Triple arthrodesis

to drop foot by shifting the centre of gravity of the foot nearer the ankle joint

(b) **LAMBRINUDI'S OPERATION FOR DROP FOOT** —The inferior surface of the astragalus is excised freely in front, so as to leave a wedged anterior part, this is introduced into a socket in the lower part of the scaphoid. By this procedure the posterior process of the astragalus impinges on the tibia and limits plantar flexion.



FIG. 437 —Astragalectomy (Whitman)

**Astragalectomy** —This operation was designed by Whitman chiefly for the correction of talipes calcaneus.

After removal of the astragalus the foot is displaced backwards and the prominence of the heel is increased. The peronei if active are transplanted into the tendo Achillis to reinforce the weakened calf muscles. The operation is being superseded by a form of triple arthrodesis (see Fig. 437).

The radiological results of operations for the various form of talipes

should not be judged upon the lateral film alone. An antero-posterior view may show an unsuspected lateral displacement of the forefoot.

### OPERATIONS FOR HALLUX VALGUS

**Excision of the Exostosis**—The simplest procedure is removal of the boss of bone on the inner aspect of the metatarsal head (Fig. 438a).

**Excision of the Metatarsal Head**—In severe cases with osteoarthritic changes in the joint the head is excised and the end of the shaft carefully



FIG. 438

(a) Excision of exostosis.

(b) Excision of metatarsal head. Too much bone has been removed and a march fracture occurred.

(c) Girdlestone's operation.

rounded. The sesamoid bones are generally left *in situ*. The operation weakens the supporting power of the foot (Fig. 438b).

**Excision of the Base of the Proximal Phalanx**—About one third of the phalanx is removed.

**Girdlestone's Operation**—The metatarsophalangeal joint is excised and the shaft and head of the proximal phalanx are removed. The base of the phalanx bearing the insertion of adductor transversus hallucis is fixed to the metatarsal head by a bone peg (Fig. 438c).

### OPERATIONS ON THE SHOULDER AND ELBOW

**Arthrodesis of the Shoulder Joint**—Two common indications are chronic tuberculous arthritis and paralytic lesions affecting mainly the deltoid muscle.

The head of the humerus and the glenoid fossa are excised down to healthy bone. The acromion process is freshened and after partial fracture is inserted into a slot in the region of the greater tuberosity to function as a graft. Fixation in abduction is maintained for several months.

The radiological evidence of bony union is generally less striking than in arthrodesis of other joints, as new bone formation in this region is scanty.

**Excision of the Elbow Joint**—This is usually performed for chronic tuberculous arthritis. A movable joint is almost the rule. Excision of the head of the radius may be done for fracture or for persistent dislocation.

## OPERATIONS ON THE SPINE

**Spinal Fusion**—The object of this type of operation is to produce fixation in a limited number of vertebræ by a bony graft in the region of the spines and laminae. The operation is most commonly performed in selected cases of Pott's disease and scoliosis.

There are two types of spinal fusion: *Albee's* and *Hibb's*.

**ALBEE'S OPERATION**—A tibial graft is placed between the split spinous processes or on a prepared bed on one or both sides of the spines.

In cases of Pott's disease the graft must extend between sound vertebræ above and below the lesion. In scoliosis very extensive grafting may be performed. This is the type of operation usually performed in England.

**HIBB'S OPERATION**—The posterior intervertebral joints are curetted. Flakes of bone from the laminae are raised and placed between adjacent laminae. Each spinous process is turned down to unite with the base of the process below.

## PART THREE

### SECTION VIII

## DISEASES OF BONES AND JOINTS OF CONSTITUTIONAL OR UNKNOWN ORIGIN

BY

DONALD HUNTER M.D. F.R.C.P., AND M. H. JUPÉ B.A., M.R.C.S.,  
L.R.C.P., F.F.R. D.M.R.E.

### CHAPTER XXXVII

#### DISEASES DUE TO DISTURBANCE OF GROWTH

#### ACHONDROPLASIA

ACHONDROPLASIA is a disease in which partial arrest of endochondral ossification leads to stunting of those bones preformed in cartilage. It therefore gives rise to disproportioned dwarfism with shortness of the limbs (micromelia). The term micromelia of course expresses nothing of the pathology, it occurs in several conditions which differ from one another clinically as well as in their pathological anatomy.

*That achondroplasia has existed for centuries is known from the statuary and pictures of ancient artists. These include two gods of ancient Egypt namely Ptah Solar and Bes. Many of the clowns of courts of grotesque figure but of normal intelligence have been achondroplastic. When young they tend to be attractive and to resemble dolls and this accounts for the fact that they were sold to the ladies of the court for large sums of money in the Middle Ages. Mentally they are normal and physically they are healthy and often very strong. Achondroplastic dwarfs are often seen in the streets and not infrequently play the parts of clowns at fairs, circuses, music halls and in cinematograph films.*

In some instances achondroplasia shows the influence of heredity. It does not seem feasible to determine the proportion between hereditary and other cases. Writing in 1912 *Rischbieth* and *Barrington* recorded 80 pedigrees in which 20 showed cases occurring in two or more generations. Their series of cases suggests that the female sex is more predisposed than the male for they found amongst 126 cases 70 females and 56 males.

**Clinical Features**—The diagnosis of the achondroplastic foetus can be made in a typical case at a glance. *The infant is one with a trunk of normal size,*

short very thick limbs head apparently rather bigger than usual with prominent frontal and parietal eminences and nose the bridge of which is depressed. The shortening affects all four limbs equally. The finger tips which in normal individuals reach to the middle third of the thigh reach in these cases no farther than the great trochanter of the femur. The shortening of bones which produces this peculiarity is greater in the proximal than in the distal segments that is it is more marked in the thigh and arm than in the leg and forearm and it affects the foot and hand least of all (Fig 439). The hands and feet are broad and thick but present peculiarities of their own. The middle digit of the hands is short—its length does not exceed that of its fellow on either side. The digits diverge from one another at their extremities forming the trident hand.



FIG 439 Photograph of an achondroplastic child aged 8 years showing the disproportion between the size of head and trunk and the limbs

The adult shows smallness of height with a normal trunk a large head and excessive muscular development. The usual height is about 4 feet. The arms are muscular and are held a little abducted from the trunk as a result of the disproportionate size of the head of the humerus. The head is brachycephalic with prominent frontal and parietal eminences. The face is relatively small the bridge of the nose being broad and flat. The teeth are normal. The normal lumbar curve is increased or is made to appear so by the excessive development of the buttocks. The genital organs are normal—the female may become pregnant and this fact makes the pelvic deformity of great importance. Achondroplasias differ from cretins and from many other dwarfs in their mental qualities. They are of average intelligence.

**Radiological Features**—Radiologically the bones of the limbs are seen to be normal in diameter in the middle though widened at the ends. They are much reduced in length and this in conjunction with the fact that all muscular attachments are exaggerated gives the impression that the cortex is widened. The shortening is met with in all bones of the limbs including the metatarsal bones and the phalanges. All bones give the impression of great strength and this is supported by the degree of muscular development. The epiphyses are normal but the ends of the diaphyses are over-developed and widened giving rise to the description that they are trumpet shaped (Fig 440). As the middle of the shaft is normal in diameter

thus widening at the extremities of the bone gives it a typical dumb bell appearance. The over-development at the ends of the bones is found wherever epiphyses are seen so that in a position such as the head of the femur or humerus, where there are three epiphyses close together the



FIG 440.—Radiogram of the arm of child shown in Fig 439



FIG 441.—Pelvis and femora of same child showing the contracted pelvis and the short wide femora. A congenital defect is seen in the lower spine and upper part of the pelvis

whole area is relatively much enlarged and the distance between the upper end of the femur and the point just below the lesser trochanter may represent one third of the total length of the bone

In a child there may be coxa valga but in the adult coxa vara is more likely. Bowing of the legs is not constantly found in radiograms. If present it may be due to angulation at the knee, and not to bending of the bones

The fibula may be shorter than the tibia and the ulna shorter than the radius. The trident hand where the ring and little fingers deviate from the middle finger, is by no means a constant feature. The vault of the skull appears too large as, owing to the defect in endochondral ossification the base is abnormally small. The bones of the base the os tribasilare consisting of the pre and post sphenoid and basi sphenoid nuclei, undergo premature fusion. This causes the base of the skull to be too short, the bridge of the nose to be depressed and the cerebellar fossa to be shallow. The angle of the clivus is more acute than usual. The mandible approximates to the normal size so that the lower jaw protrudes. The sella turcica is normal in size and shape.

The spine tends to be straight and the vertebræ to be unaltered but occasionally they are wedge shaped in which case there is kyphosis. The inlet of the pelvis is contracted partly because of under-development of the pubic and ischial bones (Fig 441). There is commonly a forward tilt of the spine on the pelvis producing lordosis. Defective development of the ilium brings the acetabulum close to the great sacro sciatic notch and throws the heads of the femora farther back than usual. It is this change which makes the buttocks more prominent and accentuates the lordosis. The epiphyses unite early.

### CHONDRO OSTEODYSTROPHY

This is a rare condition showing irregularity and fragmentation of the epiphyses of the long bones retardation of growth and some softening of the bones. It was described by *Morquio* in 1928 and in the following year *Brailsford* published very full details of a case. The pathogenesis is not fully known and histological studies are lacking. It has been suggested that it is either due to error of growth with degenerative changes in the cartilage or to some disturbance of lipoid metabolism lipogranulomatosis. The disease which is often familial is characterised by under-development, weakness of the limbs and back, painless enlargement of the joints and deformities consequent on osteoporosis and softening of the bones. It is usually seen in children and young people under the age of 20 years. The mentality is unimpaired.

**Radiological Features** —In severe cases all the epiphyses may be fragmented and deformed. They appear late and develop slowly. The shafts of the bones are widened and shortened especially at the diaphyseal ends which may be irregular. The bones are deficient in calcium. These features are well seen in the metacarpal bones and phalanges. *Brailsford* points out that the presence of large gaps between the bony extremities at the joints is an important feature and by measurement shows that the joint spaces may be more than twice the normal. Osteoporosis and pressure deformities occur. Joint surfaces such as the glenoid fossa and acetabulum are flattened and irregular. In the spine the vertebral bodies may develop unevenly so that



there is variation in size and shape of individual bones and lack of alignment of the vertebral column. In the lateral view it is common to find a defect in the upper margin of some of the bodies, and thus gives the impression that the lower margin projects forward to form a hook. The skull is usually large but normal, though there may be evidence of pressure deformity if osteoporosis has been severe.

The disease may vary in severity and in distribution, in some cases being confined to one region.

Gargoylism appears to be an allied disease, in that bone changes identical with chondro osteodystrophy are encountered. It was first described by Hurler, and is referred to as chondro osteodystrophy of the Hurler type, but the name 'gargoylism,' which is more commonly used, well describes the large head and grotesque faces of the children afflicted.

The condition is congenital and often familial and shows some similarity to amniotic familial idiocy. The causation, however, is not yet known.

Ellis has reported a number of these cases which show the 'gargoyle' face and large head. The bone deformities are identical with those described by Brailsford, Morquio and others under the name of chondro osteodystrophy. The chest in some cases is funnel shaped. The liver and spleen are usually enlarged. All are dwarfs and show varying degrees of imbecility. In two of the cases reported by Ellis, encephalography showed internal hydrocephalus and cortical atrophy.

### DYSCHONDROPLASIA

Under the heading dyschondroplasia may be grouped two conditions, both of which are rare. They are *hereditary multiple ossifying enchondromata* or *diaphyseal aclasia*, and *multiple chondromata*, which includes enchondromata and echondromata. Ollier's disease is taken to be a variation of this latter group. These conditions are fully considered in Chapter XLV.

### OSTEOPORIKILOISIS

*Osteoporikilosis* was originally described by Albers Schonberg in 1915, it must not be confused with osteopetrosis another disease first described by him. The disease shows areas of scattered density confined to the ends of the long bones, these areas are round or lenticular a few millimetres in diameter and a few centimetres in length. The size and shape of the affected bone are not altered (Fig. 442).

*Porroene's disease* has been described once only, and shows radiating or parallel lines of density at the ends of the bones. Although the appearance is somewhat similar to Ollier's disease, the bone is not altered in size and shape and the areas of translucence are absent.

At times a dense localised dendritic shadow is seen at the end of a bone. Only one bone is affected, it is possibly due to calcification in an *intramedullary haemangioma*.

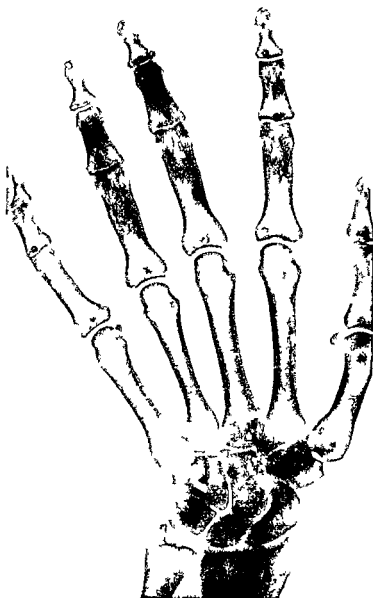


FIG. 44<sup>b</sup>—Scattered areas of density at the ends of the bones of the hand and wrist and shafts of osteopetrosis found by chance during examination of the patient after an injury.

### OSTEOGENESIS IMPERFECTA

**Clinical Features**—This condition was first clearly defined by *Lobstein* in 1833. He described the postnatal type of the disease and gave it the name *osteopetrosis idiopathica*. In 1849 *Volz* described the pre-natal type of

the disease, in which multiple fractures occur in utero and the child is often born dead. In both types the basic defect appears, from histological examination, to be an inability to form osteoblasts. There is no evidence whatever of



FIG. 443.—Multiple fractures of long bones and ribs in an infant a few days old. Many of the fractures must have been intrauterine, as repair is taking place.

vitamin deficiency. No abnormality in the serum calcium, plasma phosphorus, or calcium output has been demonstrated. The plasma phosphatase tends to show a raised value, but this is not constant.

## CHAPTER XXXVIII

### DISEASES DUE TO DISTURBANCES OF NUTRITION

#### RICKETS

**Clinical Features**—Rickets has been recognised for about 300 years and has been treated by cod liver oil for almost 200 years—a discovery made by fishermen on the shores of the Baltic Sea. During that time nobody had any idea why cod liver oil acted as a cure. Great light was thrown on the whole problem when in 1918 it was shown that puppies developed rickets on diets deficient in calcium salts and fats. In 1929 vitamin D was isolated from cod liver oil, and in 1930 it was obtained in pure crystalline form. Since its potency is in the calcification of bone, vitamin D is now called calciferol. In 1919 the ultra violet energy from the sun or from special lamps was found to cure rickets. In 1924 the reason for this was discovered, namely that calciferol is synthesised in the skin by the action of ultra violet irradiation. There are therefore two available sources of vitamin D—one in the food we eat and the other in the rays of the sun which reach our skin.

Morbid anatomists are agreed that in rickets and osteomalacia the essential abnormality is the same, namely a deficient calcification of osteoid tissue. This deficiency is generalised throughout the skeleton. In rickets, since endochondral ossification has not ceased, the zone of provisional calcification is affected too, and endochondral ossification is irregular. The nature of the calcium deficiency has given rise to much controversy, but even as long ago as 1885 morbid anatomists held the view that the calcifying mechanism which should convert osteoid tissue into true bone was deficient. Not only does any bone added by advancing endochondral ossification remain osteoid, but so also does all new bone added to trabeculae which were fully calcified at the onset of the disease. The normal physiological resorption accompanies this apposition, and consequently portions previously hard become ever softer and more liable to bend. There is no evidence whatever that bone once calcified is converted into osteoid tissue by loss of calcium salts. Under treatment by means of cod liver oil or ultra violet irradiation, ossification begins to proceed normally in osteoid tissue.

**Radiological Features**—Though rickets and osteomalacia are essentially identical, there is one great difference radiologically. Rickets affects the growing bones, while osteomalacia is found in the adult after union of the epiphyses has occurred. Rickets is therefore best seen where growth is taking place and the wrists, ankles, and knees are convenient for examination. The

anterior ends of the ribs should also be examined. Ante natal rickets is not seen in this country, but *Maxwell* has proved that it occurs in the babies of osteomalacic women in China. In this country rickets is found from birth until puberty. After that time the terms *adolescent rickets* or *osteomalacia* are used.

The cases which arise in infancy are those which concern the radiologist. The principal changes occur in the metaphyses, and the earliest alteration is a loss of definition due to slight lack of calcium salts. This increases as the disease progresses, until there is a definite area devoid of calcium salts lying between the slightly ragged end of the diaphysis and the normal epiphysis. In the more advanced cases there is a general lack of calcium salts in the bones, recognised by diminution in the cortical shadow and accentuation of the bone pattern. In very young children, owing to the absence of marked trabeculation at that age, the bone may be hazy.

The typical "saucer" appearance at the end of the growing bone is due to the absence of calcification at the metaphysis, combined with the child's movements throwing a stress on the joint. Owing to the defect in the bone forming activity of the periosteum the edges of the bone are not strong enough to prevent it from splaying. The saucer appearance is described so well by its name that no addition is needed, it is not well seen in the child who is resting in bed (Fig 445). The epiphysis itself is unaltered.

Healing is instructive. The zone of provisional calcification gradually reappears, the change being most rapid at the point nearest the epiphysis (Fig 446). Deformity is common in the region of the epiphysis, but if there is great lack of calcium in the bone, the whole shaft may be bent. If these cases are treated, a zone of thickening will form on the concave surface, forming a buttress and giving added support at the area of greatest weakness. In the hip coxa vara is common. Usually the pelvis is generally contracted, but at times it may be trifoliate. This latter is obviously due to the weight thrust of the femoral heads and sacrum.

Kyphosis and scoliosis are common sequelæ. In the skull the fontanelles

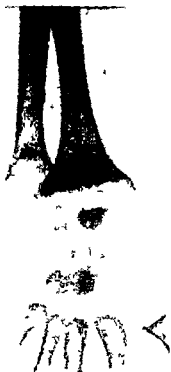


FIG 445 — "Saucer" deformity of the lower end of the radius and ulna in a patient suffering from florid rickets.



FIG 446—Rickets after treatment in a child aged 11 months. The zones of provisional calcification have reappeared. The transverse line of density near the end of each bone marks the stage of growth reached at the onset of the disease. There is deformity at the lower ends of the bones of the leg.

remain open for a long time and the bones are thin. In advanced cases the lack of calcium salts in the bones will at times cause softening so great that the base tends to be thrust up into the vault and the occiput sags over the neck giving an appearance similar to that seen in patients suffering from osteogenesis imperfecta. The anterior ends of the ribs show the saucer formation while the ribs themselves are often deformed partly owing to softening of the bone, and partly secondary to the spinal deformities. Fractures of the long bones occur. The diagnosis is not as a rule difficult in the presence of the saucer appearance but it must be borne in mind that scurvy and rickets may be seen in the same patient. Certain cases of renal rickets cannot be differentiated from rickets due to vitamin D deficiency.

### OSTEOMALACIA

In England to-day osteomalacia is a rare disease. Two types are found: the first is due to a diet deficient in vitamin D and calcium salts and may be referred to as dietetic osteomalacia; the second is due to deficient absorption and is seen in idiopathic steatorrhoea.

#### Dietetic Osteomalacia

This is endemic over wide areas in Northern India, Japan and Northern China and occurs sporadically in the Rhine Valley, Danube Valley, Vienna

and certain parts of Italy, Switzerland, Flanders, and the Balkans. It pre-eminently affects women, and is likely to recur earlier and with greater severity in each successive pregnancy. However, it is a mistake to suppose that pregnancy is essential in the aetiology; it is sometimes seen at puberty, and is quite well known to occur, though it is rare, in boys and men.

Pain occurs, especially in the back and thighs: it is aching in character and is worse in the winter months. The pelvis, thorax, or long bones show deformity in a haphazard way; one woman suffers in the pelvis, another in the ribs, and a third in both. Besides the characteristic and well-known change in the pelvis, marked deformities occur in the chest and spine. Severe kypho-scoliosis may reduce the height by several inches, and cause the head and neck to sink downwards and forwards on to the chest. Deformities of the sternum and ribs may give rise to marked prominences and depressions in the chest wall.

Coxa vara and irregular curves in the long bones are less common. The bones are soft and flexible, rather than fragile, so that bending is much more common than spontaneous fracture, though both are well recognised. The patient develops a characteristic waddling gait, and muscular weakness may add to her incapacity. In many cases the pelvic deformities interfere with marital relations or with labour, Cæsarean section frequently being necessary.

Pathologists, chemists, and clinical workers are agreed as to the essential identity of rickets and osteomalacia. What difference exists is merely that of age incidence. Osteomalacia is adult rickets. In the industrial towns of England to-day rickets is still a common disease. Morbid anatomists agree that in rickets and osteomalacia the essential abnormality is a deficient calcification of osteoid tissue. This deficiency is generalised throughout the skeleton. In 1885 Pommer stated that in both diseases the broad osteoid seams were due to deficiency of the calcifying mechanism which should convert osteoid tissue into true bone.

The experimental studies of Mellanby in 1918 and 1919 constitute the discovery of the cause of rickets and osteomalacia. He produced rickets in puppies by deficient diets, and cured it by the use of fat containing the anti-rachitic vitamin. Meanwhile the anti-rachitic power of ultra-violet irradiation was discovered by Huldshinsky in 1919. In 1924 it was discovered that certain foods developed anti-rachitic powers when exposed to ultra-violet irradiation. It has since been possible to cure rickets in children by feeding them on irradiated foods. It is the sterols in such foods that can take on anti-rachitic potency, in particular ergosterol, for vitamin D is a derivative of this substance. In 1930 vitamin D was isolated in pure form by extracting ergosterol from yeast and irradiating it in alcoholic solution as it passes through quartz tubes. The pure vitamin is called calciferol, from its power

to induce calcification in tissues especially in osteoid tissue. It is 300,000 times as potent as cod liver oil weight for weight.

The blood chemistry is comparable in experimental rickets of rats in children with rickets and in women with osteomalacia. The plasma phosphorus and sometimes the serum calcium are diminished. Adequate treatment restores the low figure to normal. In the absence of vitamin D the utilisation of calcium and phosphorus is impaired. The low blood phosphorus or calcium is promptly restored to normal by the use of cod liver oil, calciferol or ultra violet irradiation. At the same time ossification begins to proceed normally.

#### Radiological Features

—The degree of lack of calcification in the radiograms will vary with the severity of the disease and it is therefore important to use controlled radiograms. In the slight cases the bones of the patient will be slightly more translucent than those of the control. The cortex will be less marked and the bone pattern will be accentuated especially the trabeculation. In the severe

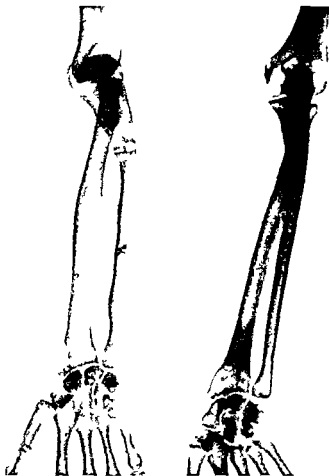


FIG 447.—Osteomalacia. Woman, aged 33 years. The lack of calcium is clearly shown when compared with the normal control. Pseudo fractures (Looser's zones) are present in the ulna.

examples there will be little or no difference in density between the bone and the surrounding soft tissue and the cortex will appear as a pencilled out line. The bone pattern will have disappeared, the long bones will bend and occasionally show fracture (Fig 447). All deformity apart from fracture is the result of weight stress or muscular action.

The pelvis has attracted much attention largely owing to the fact that the



disease is often first noticed during pregnancy. It shows practically any pressure deformity, but chiefly it is trifoliate, owing to the thrust of the heads of the femora and the sacrum (Fig 448). In the spine lordosis is marked largely on account of the forward displacement and downward rotation of the sacrum. There is usually kyphosis, in some cases so great that the lower ribs overlap the iliac crests. The chest and ribs are usually deformed in severe cases. The vertebrae are biconcave and have the appearance of fish vertebrae (Fig 449). It must be remembered that this finding is common to many



FIG. 448.—Same patient as Fig. 447. Deformity of the pelvis is present due to the thrust of the femoral heads. The rami of the pubic bones have been severed as a result of the crushing.

diseases where calcium deficiency plays a part. In extreme cases the vault of the skull may show numerous areas of uneven translucence varying in size and shape but all fairly clean cut, just as they are in myelomatosis (Fig 450). *Brailsford* has published a case of renal rickets showing these appearances. If the bones of the skull become very soft the vault may tend to sag over the neck, giving the 'tam-o'-shanter' appearance.

Pseudo fractures have been pointed out in osteomalacia, and they are well known in China. They appear as areas of complete translucence running across the bone, the edges being quite clean cut and separated from each other



FIG. 449—Same patient as Fig. 44. There is extreme lack of calcium in the bodies of the lumbar vertebrae which have become biconcave.

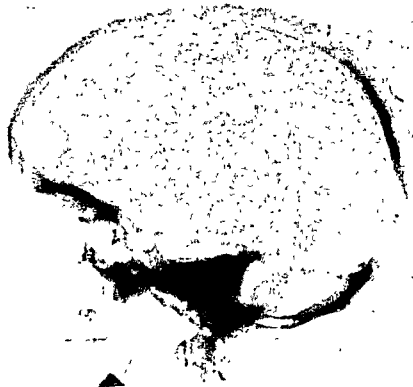
by 1 or 2 mm (Fig. 44). These zones are often referred to as *Looser's zones* and *Looser* describes that on microscopic examination the old laminated bone disappears by lacunar absorption being replaced at the same time in the marrow by mottled bone devoid of calcium to begin with" (*Köhler*).

#### Idiopathic Steatorrhœa

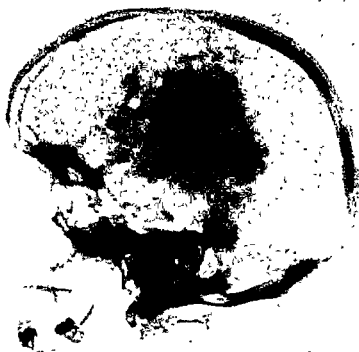
The other cause of osteomalacia is idiopathic steatorrhœa (*Celiac disease* or *cœliac riclets*). This disease occurs in both sexes and the history nearly always goes back to early childhood. The following features may be present: fatty stool, dilatation of the colon, tetany, osteomalacia, anaemia, skin lesions, and infantilism. These

manifestations develop in spite of an adequate diet. We must therefore suppose that there is some disturbance of gastro-intestinal function resulting in deficient production, absorption, or utilisation of one or more essential factors. In 15 cases investigated in 1932, steatorrhœa and disturbances of calcium metabolism were alone common to the whole group. In 13 cases out of the 15 the serum calcium was low. The plasma phosphorus was low or normal in 10 cases; in the remaining 3 it was above the limits of normal. Calcium balance estimation showed high figures for faecal output and very low figures for urinary output. Changes in the skeleton were found in all cases investigated.

Radiograms of the bones show the diminution of density and deformities of osteomalacia (Fig. 451). A barium enema will show the dilatation of the colon



(a)



(b)

FIG. 450.—Same patient as Fig. 447. (a) Scattered areas of translucence are seen in the skull.  
(b) After six months' treatment these areas have almost completely disappeared.

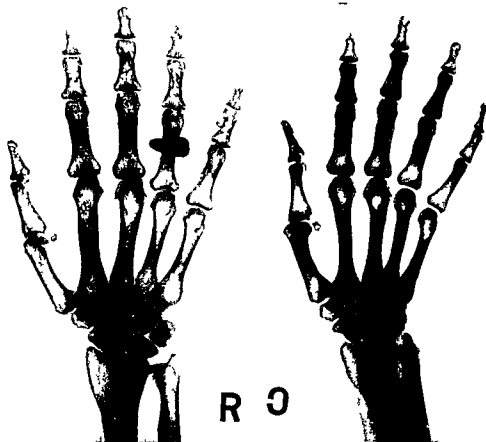


FIG. 451.—Idiopathic steatorrhoea in a female aged 23 showing the deficiency of calcium in the bones compared with the normal control.

### SCURVY

Vitamin C has been chemically identified, synthesised and named ascorbic acid. It can now be manufactured from starch and sold for a shilling a gramme. Vitamin C is destroyed by heat and for this reason dried milks must not be used as infant foods without the addition of orange juice. Artificially fed babies sometimes suffer from scurvy but it is practically never seen in a breast-fed baby. A child is unlikely to suffer from infantile scurvy before 8 months or after 2 years of age. Bachelors' scurvy is still occasionally found among adults who live in poverty. Sometimes the disease comes on in a patient under treatment for gastric ulcer when the diet is deficient in fresh fruit and vegetables.

Scurvy is characterised by changes in capillary endothelium leading to hæmorrhages and by diminished activity of hæmopoiesis. Thus anæmia may

occur apart from hæmorrhage. In infantile scurvy the bones are affected. In addition to subperiosteal hæmorrhage there is cessation of osteogenetic activity, arrest of endochondral ossification, but no deficiency in the calcification of the matrix of cartilage. These changes result in osteoporosis, sometimes with spontaneous fracture.

**Radiological Features**—Radiologically there is little difficulty in recognising a marked case of the disease. In less severe cases it is more likely to be overlooked than to be incorrectly diagnosed. The limb involved may be acutely painful, so that periodically infants are sent to be radiographed with a diagnosis of pulled elbow or injury to the leg, and are found to be suffering from scurvy. The most convenient joints to examine are the wrists and ankles, but the commonest positions in which to find subperiosteal hæmorrhages are the lower ends of the humerus and femur. The anterior ends of the ribs give a characteristic picture and should always be examined. The earliest sign in a radiogram is loss of density and pattern of the cancellous bone, so that this region becomes homogeneous and hazy (Fig. 4a2).

The cortex is thin and seems more obvious than usual. This gives rise to the apt description that the bone is seen as a ground glass area surrounded by a pencilled outline. It also accounts for the epiphysis appearing as a ring of cortex with a structureless centre. In more advanced cases an irregular line of relatively dense calcification forms at the epiphyseal end of the shaft. This is the zone of debris, the *Trummerfeld* one, or the *white line* of *Fraenkel*. It is actually an excessively thick, irregular zone of provisional calcification, for the provisional changes, including calcification of the cartilage matrix, are not inhibited in vitamin C deficiency. To the metaphyseal side of this dense zone is a zone of relative translucence called the *Gerust mark*—one of *Lehndorff*. This is due to partial arrest of osteoblastic activity, a direct effect of vitamin C deficiency. It is at this area of weakness that the bone tends to give way. Its width indicates the length of time that the condition has been present, and the severity of the disease.



FIG. 4a.—Scurvy in a child aged two years and eight months. Lack of density and loss of bone pattern is apparent, especially in the epiphyses. The appearance of crushing at the ends of the shafts is seen.

There may be slight lateral displacement of the epiphysis and the adjoining dense zone at the end of the shaft (Figs 452 and 453) Bones showing such displacement heal without deformity and need no treatment to correct this apart from the correction of the dietetic error

Just as the shaft shows an area of increased density at the epiphyseal junction, so may the epiphysis show a ring about its centre This is called *Ilmberger's sign* and appears as an area of irregular density about the central

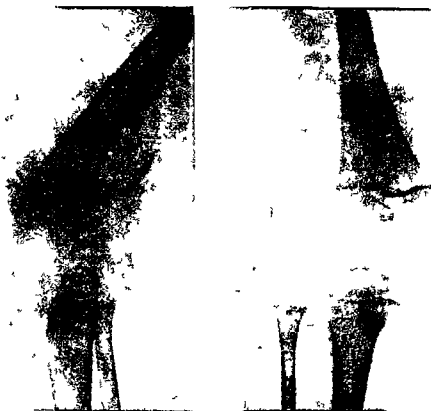


FIG 453 —Scurvy in child aged 10 months Showing subperiosteal hæmorrhage and the lateral spur formed by the bone at the end of the shaft giving way

nucleus of the epiphysis which is more translucent than usual and gives the ground glass picture described above This is best seen in the bones of the carpus and tarsus, especially the os calcis and astragalus It is not a sign on which great stress can be laid, it probably corresponds with the lines of arrest of growth due to many causes which are seen in the long bones These translucent areas in the epiphyses are seen as long as five years after the disease has been arrested In advanced cases when the translucent zone of the shaft has given way, the end of the bone gives an impression that it has been crushed or

impacted. This crushing sometimes with displacement, gives rise to a shadow like a spur at the diaphyseal extremity. This is referred to by American authors as the *lateral spur of Pelkan*. It is not a true spur, but is simply the result of the weakness of the translucent osteoporotic area and slight displacement of the dense area.

Subperiosteal hæmorrhage may show itself in two ways. If the bleeding is recent and considerable the periosteum will be stripped from the bone and may be seen as a fusiform area of very slightly increased density with a thin linear outline. If the hæmorrhage is of longer standing it may be partially



FIG. 434.—Scurvy in a child of eleven months. The expansion at the ends of the ribs is seen in addition to the changes at the upper ends of the humeri.

or wholly calcified and will then show as a dense shadow surrounding the bone or joint (Fig. 433). The anterior ends of the ribs give great assistance in some cases: the tips become spatulate and expanded to almost twice their usual size. The extremity is round and easily distinguishable from the saucer shaped end of the rachitic rib (Fig. 434).

THE DIFFERENTIAL DIAGNOSIS is not always easy and congenital syphilis would give rise to difficulty were it not for the different age incidence. In the latter disease the periostitis and the irregular density at the ends of the bones are confused with the subperiosteal hæmorrhage and dense zone of scurvy. Congenital syphilis occurs in the first few weeks of life,

and its manifestations are usually bilateral. Scurvy may attack more than one bone but it is by no means symmetrical, and the hæmorrhages are fusiform whereas syphilitic periostitis tends to raise the periosteum evenly and uniformly. The dense area of congenital syphilis is more uneven than that of scurvy, and the bones show no "ground glass" texture. *Rickets* should not be confused with scurvy in the presence of the "saucer shaped" ends of the shafts of the bones. The dense zone of scurvy and the deformity of the ends of the bones should leave no difficulty. The ends of the ribs should be examined if there is any doubt. *Poisoning by lead and phosphorus* and *healed rickets* show very clean cut areas of density, which are characteristic. In these conditions there is no alteration in the bone structure. In the *marble bone disease of Albers Schonberg* the widening of the ends of the bones, absence of loss of calcium, regularity of the zone of density and absence of the zone of decreased density will decide.



## CHAPTER XXIX

### DISEASES DUE TO DISTURBANCE OF METABOLISM

#### GOUT

**Clinical Features**—Gout is a constitutional disease in which joint symptoms are dominant. Its evolution is usually characteristic. The first attack occurs after the age of 40. Its onset is sudden; it generally lasts only three to seven days and then disappears completely. A large toe or with almost equal frequency another joint may have been affected—an instep, heel, ankle or knee. After a year or two another attack appears, often more severe and of seven to fourteen days' duration. It too disappears, leaving the joints normal. Sooner or later the disease increases in severity and attacks occur every few months. At first attacks are monarticular; later attacks may be polyarticular, another region being affected as the first recovers. After a variable time the fairly short attacks of acute arthritis disappear, leaving the patient normal. Many years—the average being twelve—after the first attack an important change occurs: the joints no longer recover completely and the disability may become chronic. Thus is initiated the second stage of gout, namely chronic tophaceous gout. In this stage the joints are liable to acute exacerbations with incomplete remissions. Finally exacerbations cease and the patient presents misshapen extremities with multiple tophi; yet the joints are relatively painless.

About 98 per cent of patients who have gout are males. In a woman the diagnosis should be made with caution, but can be based on the same features as those of gout in males. Acute gout is the commonest form of acute arthritis among men over 40 and should always be borne in mind after excluding gonorrhoea and acute trauma. Although attacks may begin at any hour, they occur characteristically between 2 and 7 a.m. and the onset and development of an attack occur with unusual speed.

No other arthritis develops so abruptly to reach its maximum within one or two days. The pain, sometimes mild or moderate, is often excruciating, beyond that of any other type of arthritis. The affected extremity is more frequently hot, bluish red and shiny than not, and contrasts with the cold, clammy, bluish white swellings of rheumatoid arthritis. Shiny skin, œdema with pitting and desquamation of the skin as the attack subsides are often notable. Acute gout is prone to appear after dietary indiscretions, but may affect vegetarians and teetotallers. An attack may be precipitated by trauma, exposure to cold, or a surgical operation.

Gout tends to involve certain extra-articular tissues, especially the skin

cranon bursa. Renal colic may occur from urate stones or gravel. The supposed high incidence of gout a century ago may have been false, but certainly to-day it is wrong to call the disease rare. Its incidence is from 5 to 8 per cent of cases attending special arthritis clinics.

**Radiological Features**—It is not until the disease is advanced that radiological changes can be demonstrated and for this reason the radiograms in the majority of cases of gout show no abnormality.

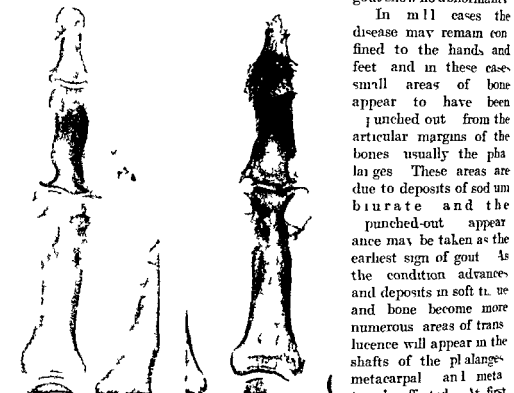


FIG. 43. (a) Normal phalanx. (b) Soft tissue shadows and flecks of calcified material. (c) Large punched-out areas with marginal bony spurs about the interphalangeal joint.

of the shaft and disorganisation of joints may result. The bone lesions, together with the tophi in the soft tissues, give rise to the fantastic deformities which result. A large uncalcified deposit may erode the bones forming a joint. Small spurs of bone are then seen at the margins of the erosion (Fig. 43c).

In certain instances the deposits both in soft tissue and in bone may undergo calcification and become flecked with shadows of calcium salts or the whole may be rendered opaque. In this way the gouty chalk

In all cases the disease may remain confined to the hands and feet and in these cases small areas of bone appear to have been punched out from the articular margins of the bones, usually the phalanges. These areas are due to deposits of sodium urate and the punched-out appearance may be taken as the earliest sign of gout. As the condition advances and deposits in soft tissue and bone become more numerous, areas of translucence will appear in the shafts of the phalanges, metacarpal and metatarsals affected. At first they are small and discrete but gradually they increase in size and number until the bones become honeycombed. Collapse

stone," common in the days of our forefathers, but now rare, may be demonstrated radiologically

**Differential Diagnosis** between this disease and *multiple chondromata and enchondromata* may be difficult but the presence of the "punched-out" areas and the fact that in gout the lesions do not necessarily arise centrally in the shaft of the bone may help. The history and clinical examination will decide. Other diseases which may cause confusion are some forms of *tuberculous dactylitis* and *osteitis tuberculosa multiplex cystica* (Boeck's sarcoid)

## THE LIPOID GRANULOMATOSES

The association of lesions in the membrane bones of the skull with diabetes insipidus and exophthalmos was first ascribed to some form of dyspituitarism but enough is now known to place it in the group of general metabolic disorders. The term "lipoid granulomatosis" is applied to a group of diseases in all of which there is a disturbance of lipoid metabolism, manifested by deposition of fatty substances in cells of the reticulo-endothelial system. As the lipoid substances approach nearer to the chemical value of ordinary fat, so the tumour masses appear more yellowish and thus are sometimes called xanthomata. Isolated xanthomata may appear in the ends of long bones and in connection with tendon sheaths and bursæ. By some writers they have been regarded as giant celled tumours and confused with *osteitis fibrosa*. The group of lipoid granulomatoses includes GAUCHEL'S DISEASE in which the lipoid deposit is *kerasin*, the NIFMANN-PICK DISEASE in which it is a *phosphatide*, and XANTHOMATOSIS SCHULLER-CHRISTIAN SYNDROME where *cholesterol* is responsible. Amyrotic family idiocy is thought to belong to this group. The histology of each presents a characteristic type of cell common to all the form cell which has a vacuolated cytoplasm, the vacuoles of which in appropriately stained fresh sections can be demonstrated to contain the corresponding lipoids. The distribution of infiltration in different organs is responsible for certain distinguishing characteristics in each disease. In all three the bones may be affected and the spleen, liver, and even lungs are possible sites.

### Xanthomatosis

In xanthomatosis the patient usually seeks advice on account of polyuria, but pain or exophthalmos may be the original cause of medical examination. The exophthalmos depends on the destruction of the bones about the orbit, especially the roof, and the diabetes insipidus on the fact that the pituitary region is invaded and the tuber cinereum involved.

The intracranial pressure may be increased and the sutures separated. The long bones may be affected as well as the flat, and in some cases the skull escapes entirely. *Snapper* has two cases where the bones have been affected but the skull has escaped so that there is no diabetes insipidus. We have

studied three cases in which bones were involved and though polyuria was present no bone lesion in the skull could be demonstrated in radiograms.

The ages of cases have varied from 2 to 55 years but it is commonest in the young. The patients if children are undersized. Clinically definite lumps are found about the bone lesions. Radiologically the skull attracts most attention and areas of bone destruction are seen most commonly in the

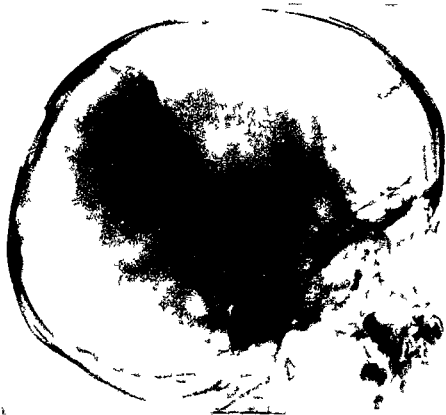


FIG. 456.—Xanthomas. Typical large map-like area of bone destruction in the fronto-parietal area with smaller areas in the temporal and parietal bones in a young child. It is of interest that the occipital bone has not escaped involvement.

fronto-parietal region and the base and especially in the vicinity of the pituitary fossa. The occiput usually escapes. Both tables of the vault are equally involved; the edges are clean cut but uneven; there is no suggestion of periosteal reaction or repair. The areas vary in size and gradually become confluent (Fig. 456) forming the map-like appearance (*carte géographique*) and making the whole skull soft and uneven (*crâne en gelée*). The involvement of the base may be clearly seen and can be recognized by the lack of calcium. The invasion of the orbits is not easy to demonstrate but can be shown in

marked cases. The maxilla and mandible do not escape and this accounts for gingivitis being an early manifestation.

The flat bones of the other parts of the body are commonly involved especially the pelvis though the long bones are by no means immune (Fig. 457). When a solitary area is examined the appearance in the early stage is usually

mistaken for a low grade infective process in which there is no local reaction that is a solitary area of erosion irregular in outline and with no reaction. As the process advances new sites are invaded and the areas become larger and may show a few strands of coarse trabeculation the edges are clean cut and irregular. They may be indistinguishable from areas of fibro cystic disease. Destruction of bone is usual but occasionally the lesions appear to undergo sclerosis and thus may have given rise to the statement that occasionally in children cases undergo spontaneous cure.



FIG. 457.—Xanthomatous. Two areas of destruction about the acetabulum and one at the lower part of the sacro iliac joint in a woman aged 33 with a history of four years polyuria and two months pain in the left hip. These areas healed completely when treated by X rays.

In the long bones and ribs fractures may occur at the sites of the lesions. The deposits may involve the reticulo endothelial system anywhere in the soft tissues but it is only in the chest that the radiologist is likely to detect them when small areas of scattered density may be seen in the lung fields.

DIFFERENTIAL DIAGNOSIS is easy both clinically and radiologically in a typical case but *neoplasm of the skull* must be excluded this difficulty is only likely to arise when the condition is confluent. In the early stages *myeloma*

*tos* must be ruled out, but in this latter condition the lesions are usually round and not irregular, the urine must be examined for Bence Jones protein.

In *renal rickets* *vitamin D* deficiency, and *osteitis deformans*, in the skull there is a lack of clarity, so that the whole appears woolly. The other

bones should give the diagnosis. In the long bones and flat bones in some cases it is impossible to differentiate between xanthomatosis and focal fibro cystic disease.

**TREATMENT**—The local lesions in xanthomatosis tend to yield to X-ray therapy temporarily.

### Gaucher's Disease

Gaucher's disease may be familial. It is characterised by splenomegaly, anaemia, enlargement of the liver, and sometimes thrombopenia. The substance deposited in the tissues is kersin—a galacto lipid. In 1922 Pick discovered a gross osseous form of the disease with angular curvature of the spine and pathological fractures. If the bones are examined all the typical features of lack of calcium are seen owing to lipid deposits. Radiologically the affected bones show osteoporosis and thinning



FIG. 458.—Radiogram of a case of Gaucher's disease taken on the same film with a normal control. The diagram illustrates the degree of widening of the lower end of the femur. (Professor Snapper's case.)

of the cortex, which may in extreme cases become expanded.

At first sight the condition is likely to be mistaken for focal osteitis fibrosa cystica affecting several bones, but there is no evidence of true cyst formation with typical areas of expansion and strands of trabeculation (Fig. 458). The diseased bones may show signs of compression, and this is best seen in the head of the femur. The lower ends of the femora may be widened evenly, and this is said to be characteristic. The skull may be involved.

**Niemann-Pick's Disease**

This rare disease was first described by *Niemann* in 1914, it is found in young children usually girls of Jewish parentage. The proportion of affected females to males is quoted as 6 to 1. The chief features are enlargement of liver and spleen and wasting. The child does not usually survive the age of 2 years. The condition is very similar in appearance to *Gaucher's disease*, and *Pick* quotes *Niemann* as saying that the primary difference depends on the early age at which the disease appears, and the rapid course. As in *Gaucher's disease* areas of rarefaction may appear in the long bones and these are found to contain a phosphatide. Widening of the femora has not been recorded.

## CHAPTER XL

### DISEASES DUE TO DISTURBANCE OF THE ENDOCRINE SYSTEM

#### GENERALISED OSTEITIS FIBROSA HYPERPARATHYROIDISM

**Clinical Features**—Generalised osteitis fibrosa is a disease which progresses with pain fractures and disabling deformities and if untreated is usually fatal. There is widespread pathological resorption affecting all the bones. In addition there are multiple foci of osteitis fibrosa with or without benign giant celled tumours and cysts. It is a disease entity distinctly different from focal osteitis fibrosa. Besides symptoms due to changes in the bones there may be hypotonia and muscular weakness anorexia sometimes with nausea vomiting and abdominal cramps polydipsia and polyuria renal calculi sometimes with colic and hæmaturia wasting in advanced cases high serum calcium low plasma phosphorus increased output of calcium in the urine and evidence in radiograms of generalised osteoporosis of the skeleton with or without deformities cysts and tumours. It is more than twice as common in women as in men and it usually appears between the ages of 30 and 50. The number of recorded cases which have been arrested or cured by removal of a parathyroid tumour is now more than fifty. This operation brings about dramatic changes. Usually the pain in the bones goes immediately. In many cases polydipsia and polyuria are promptly abolished often there is disappearance of gastro intestinal symptoms gain in weight and strength and ability to resume work. Decrease in size of osteoclastic tumours of bone within a few weeks of operation on the neck has now been many times recorded and as a rule the level of the serum calcium and plasma phosphorus and the excretion of calcium in the urine are restored to normal.

**Radiological Features**—Radiologically the most striking difference between this and the focal type is the great lack of calcium throughout the whole skeleton so that extensive deformity often results and the bones may break. The cortex loses its ivory character and may be reduced to a thin uneven linear shadow. The medulla becomes less dense with resulting accentuation of bone pattern at first but later the cancellous bone tends to fade entirely. The skull shows a finely mottled appearance with small areas of bone of normal density surrounding areas of relative translucence. This is well seen in the outer table. Pale cyst like areas may be seen scattered about the calvaria. The vertebræ may be biconcave like fish vertebræ and they may be reduced in height. The cysts are similar to those seen in focal osteitis fibrosa except that they are often smaller and usually cause less expansion of the cortex. At



tunes the cysts may be difficult to see, so great is the lack of calcium in the surrounding bone. The cystic changes occur late in the disease, and their presence is not essential to the diagnosis (Fig. 459).

All the bones of the body may be affected, even the phalanges of hands and feet (Figs. 460a and 460b). The pelvis shows pressure deformities due to the thrust of the femoral heads. Cyst-like areas are common in all the pelvic bones. The femora and ribs may bend and collapse. The excessive excretion of calcium may give rise to calculi in the urinary tract and this should therefore be examined. On radiographing the chest and neck it must be borne in mind that a parathyroid tumour may vary greatly in size and position. In more than one case its shadow has been found in the mediastinum. Lack of density of the bones should make it clear that the case is not one of focal osteitis fibrosa, and therefore controlled radiograms should always be taken.

**Differential Diagnosis** — The chemical findings are the deciding factor in the differential diagnosis. In certain cases it is difficult to differentiate between hyperparathyroidism with few and inconspicuous cysts and osteomalacia. A careful search must be made for cysts and the chemistry investigated. In *osteitis deformans* the radiological picture is definite and the blood chemistry normal. *Multiple myelomatosis* may cause difficulty, owing to widespread osteoporosis, the serum calcium is usually normal but is occasionally raised.

The progress of the disease should be checked radiologically after removal of a parathyroid tumour. In certain instances a further tumour may be present, and may only be detected after it has been found that the removal of the first has not brought about relief of symptoms and restoration of the chemistry to normal (Figs. 460a and 460b).



FIG. 459.—Hyperparathyroidism. Cyst formation and poor calcification in the bones of the forearm compared with a normal control.



FIG. 460 (a)—Hyperparathyroidism. Extreme osteoporosis compared with the normal control with thinning and bending of the bones in a woman aged 41. Note the large cystic area in the middle metacarpal causing pressure deformity of the neighbouring bones.

### THYROTOXIC OSTEOPOROSIS

It is of course known that thyroxine raises the basal rate of combustion of carbohydrate fat and possibly protein consequently appreciably increasing the total heat production of the organism. Its effect however on inorganic salt metabolism has been worked out only recently. In cases of thyrotoxicosis the serum calcium and plasma phosphorus figures are invariably normal but the calcium excretion may be raised as high as eight times the normal. In some of these cases extensive osteoporosis of bone occurs by the mechanism of osteoclastic lacunar resorption. The absence of calcium is generalised and gives a picture of loss of density with thinning and pitting of the cortex and

accentuation of the trabecular pattern. In our experience controlled radiograms of the bones in thyrotoxicosis reveal poverty of calcium in less than half the cases examined (Fig 461). In cases of hyperthyroidism with spontaneous fracture there may be radiographic evidence of osteoporosis not only in the

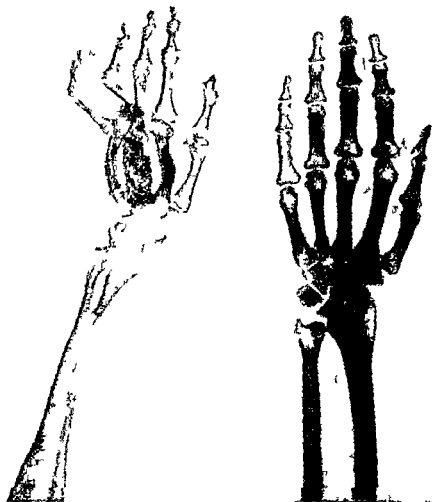


FIG 460 (b)—Hyperparathyroidism. Same case as Fig 460 (a) 18 months after removal of parathyroid tumour. The density of the bones has almost completely returned and the cysts are disappearing.

fractured bone but also in all other bones examined. The process may at times be uneven and *Snapper* has had a case where this unevenness is very marked. During treatment with iodine and after subtotal thyroidectomy, as the patient improves and the basal metabolic rate approaches the normal the calcium excretion also returns towards normal. The density of the radiographic



FIG. 461. Thyrotoxic osteoporosis in a girl aged 19 years who suffered from severe hyperthyroidism with enlargement of the gland for a number of years (with control).

shadows of the bones may then improve. However, partial thyroidectomy does not necessarily ensure this.

### INFANTILISM

It is to be noted that dwarfism may occur in a person who is by no means mentally or sexually infantile. This is the case, for example, in achondroplasia. It is important also to recognise that pygmy races form an example of physiological dwarfism, the best known being that pygmy tribe of the Belgian Congo. Infantilism implies dwarfism with, in addition, the persistence of infantile characters and a general retardation of development, bodily, mental, and sexual. It is impossible to make a satisfactory classification of the causes of infantilism, since we know so little as to the factors responsible for normal growth. In many cases no cause is evident. In others, the failure in development has followed some definite disease, and lastly, there are

cases of direct endocrine origin. Syphilis, chronic nephritis, alcoholism, lead poisoning, congenital heart disease, malaria, and hookworm disease may all cause delay in development and in the onset of puberty.

**Lorain Type of Infantilism**—In 1866 *Shaffhausen* of Bonn described a dwarf, 61 years of age, who measured  $37\frac{1}{2}$  inches, had the face of a child, bilateral cryptorchidism, and ununited epiphyses. The permanent teeth had erupted at 22 years of age. In 1871 *Lorain*, in connection with infantilism occurring in the children of tuberculous parents, gave the following description of the syndrome, which now bears his name. A slender, fair, well-proportioned figure of adolescence, before the changes of puberty have occurred, the legs being long out of proportion to the trunk, and the epiphyses delayed in junction. The features are well drawn, and there are no abnormal deposits of fat. The skin and hair are smooth and fine, the hands and fingers delicate. The genitals are underdeveloped, and the secondary sexual characteristics are often absent. The intelligence is usually not impaired. The voice remains infantile. The teeth erupt late, and the permanent teeth are sometimes still unerupted at 25 or even 30 years of age.

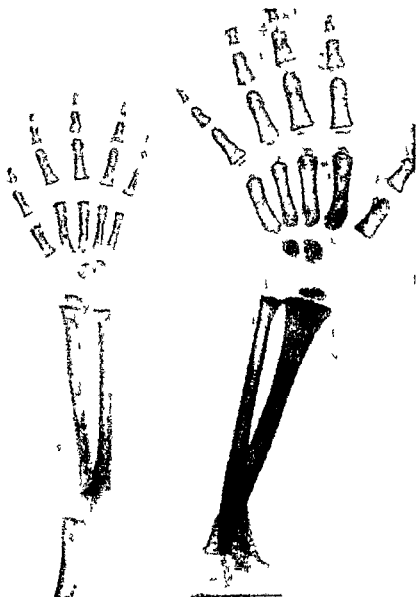


FIG 46'—Infantilism of the Loran type in a boy aged 2½ years. The bones are very small compared with those of the normal control and the epiphyses of the metacarpals and phalanges have not yet appeared.

It is this condition which was afterwards called *atelerosis* by *Hastings Gilford*. The cause of the syndrome is unknown. Several cases may occur in one generation. Such dwarfs are often seen on the stage. They are sometimes very intelligent and their mentality is then very interesting. *Walter de la Mare*, who knew one intimately, has written about her in his

*Memoirs of a Midget* They are usually though not always sterile. An ateleiotic mother has been known to give birth to a premature child which ultimately grew to normal adult size. In 1919 *Simmonds* of Hamburg found atrophy of the anterior lobe of the pituitary gland in a dwarf of the Loran type who belonged to a performing troupe. The posterior lobe was histologically normal but the anterior lobe was almost entirely replaced by fibrosis and cyst formation. He supposed on evidence which seems inadequate that the condition was due to embolism. Similar features may be seen in patients suffering from suprasellar craniopharyngioma. Usually in these cases the subject does not show such definite retardation of growth and there will be clinical signs of an intracranial lesion. The subject is fully dealt with in Chapter IV.

**RADIOLOGICALLY** the bones appear normal in all respects except size. The epiphyses appear late (Fig. 462) and union is delayed in varying degree. In many cases union never takes place. The sella turcica is within the limits of normal. The skull conforms to the childish shape with prominent forehead and parietal eminences. The sutures including the basisphenoid remain open. The teeth are erupted late. If a suprasellar tumour is present the pituitary fossa will be enlarged or suprasellar calcification may be present.

**Cretinism**—Endemic cretinism occurs wherever goitre is prevalent. It is a congenital thyroid deficiency appearing in the child of a goitrous mother. It differs from sporadic cretinism in the fact that the patient usually shows a goitre. Sporadic cretinism should be suspected in a child overweight at birth. Otherwise it is rarely recognised before the child is 6 months old. It is noticed that the patient does not grow so rapidly and is not bright mentally. The face is large and bloated, the eyelids are puffy and swollen, the alæ nasi are thickened and the nose is depressed and flat. The tongue looks large and hangs out of the mouth. Dentition is delayed. The hair may be thin and the skin dry. The abdomen is swollen, the hands and feet are ungainly and the face has a waxy sallow tint. The fontanelles remain open and there is muscular weakness. In the supraclavicular region are large pads of fat. The child shows poor mental development and in some cases is a complete imbecile.

**RADIOLOGICALLY** in the endemic cases the bones are short and thick and the epiphyses appear late and tend to be irregular and deformed. They are often fragmented. There is delayed union. The base of the skull is abnormally short and the maxilla is prominent. The total width of the skull is greater and the bones thicker than normal. There is delayed ossification of the vault. Wormian bones are frequently seen. The sella turcica may be large. The vertebral bodies are reduced in height. Deformity of the hip may take the form of coxa vara or coxa valga.

Untreated cases of sporadic cretinism are very rarely seen in this country and so observations must be limited in the few cases of young children and

infants which we have seen, a band of extra density, similar to that seen in lead poisoning, may occur at the growing ends of the bones. This band varies in width and density and disappears slowly when treatment is given. It is possible that this is the converse of the osteoporosis seen in hyperthyroidism. Presumably, unless treatment is given, the density will increase from birth onwards, so that the bulk of the bone will become extra dense (Figs. 463*a* and

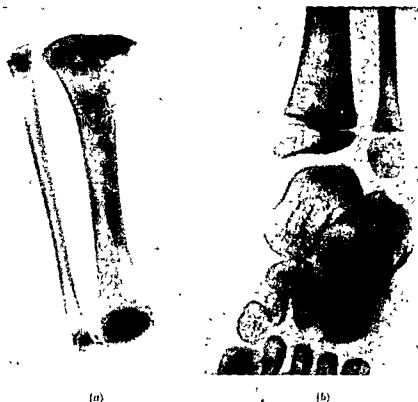


FIG. 463.—(a) Cretin (untreated) at the age of  $1\frac{1}{2}$  years showing the zone of increased density at the ends of shaft. (b) The same child at age of 4 years and after adequate thyroid treatment.

463*b*). The conditions for which it is likely to be mistaken are lead poisoning, osteopetrosis, and hypervitaminosis D. The general appearance of the child is the best guide to the diagnosis of cretinism.

**Mongolism.**—This condition derives its name from the appearance of the child. The eyelids are slanting and the eyes wide apart, the face is broad, the hair straight, the skin sallow, and the mouth open. In stature the child is small and usually broad, with a mentality well below normal. The condition is congenital, and the child affected is usually born to parents who are advancing in years. The diagnosis is self-evident. The cause is unknown.

**RADIOLOGICALLY** the bones are seen to be short for the age of the child ossification is delayed. The chief changes are seen in the hands. The thumb is often short and the middle phalanx of the fifth finger is short and curved. It is not uncommon to find epiphyses at both ends of the metatarsals and metacarpals. In the skull the sutures remain ununited for an unusually long time.

**Renal Dwarfism**—Chronic nephritis beginning in early childhood is sometimes associated with dwarfism. In such cases dwarfism may exist alone or may be complicated by rickets with the corresponding enlargement of the epiphyses, beading of the ribs and genu valgum. The mechanism producing the bone deformity is not understood. The renal defect causes phosphate retention which progresses with the rise in blood urea. As

the plasma inorganic phosphorus rises the serum calcium falls and defective bone apposition results. The age of onset is in late childhood up to puberty. Polydipsia and polyuria may be present and albumin is found in the urine. Cardiovascular hypertrophy is slight or absent. The disease is always fatal.

*Parsons and Teall* describe two types of skeletal change—rachitic and woolly or stippled. Deficient calcification of the bones is common to both types but is more marked in the woolly type. Wrists, ankles, knees and skull should be examined.

**IN THE RACHITIC TYPE** the changes resemble those of rickets but the saucer formation is not so marked and the ends of the bones are not so splayed. One of the earliest signs is a slight widening of the space at the epiphyseal junction (Fig 464). As the condition advances the end of the



FIG 464—Renal dwarfism. The rachitic type in a boy aged 14 years.

diaphysis becomes ragged and the space widens still more until the appearance approximates to that of ordinary rickets. The epiphyses are delayed in appearing and uniting. The skull shows no changes apart from delayed closure of the sutures.

**THE WOOLLY TYPE** is unlike any other disease in its appearance, especially at the growing ends of the bones. There is lack of calcification and the trabeculae are more obvious than usual. The metaphysis shows uneven ossification.



and loss of definition; hence the terms "woolly" and "stippled." The bone just under the periosteum in the region of the metaphysis often casts no shadow at all, and this is aptly described by *Trall* as resembling a post rotting in the earth. If this process be confined to one side of the bone, the shaft will tend to collapse and close up the defect, thereby giving a very typical deformity. We have seen this affect the whole width of the bone, so that the epiphysis, with part of the diaphysis, becomes separated (Fig. 465). Fractures of the shaft may occur. The changes in the skull resemble those of osteitis deformans, with thickening and separation of the tables of the vault and scattered areas of uneven density (Fig. 466). The base appears to be thickened to a greater extent than in osteitis deformans, in which the vault is chiefly affected. Osteitis deformans, of course, does not occur in children.

*Ellis* and *Evans* have shown that, in addition to chronic nephritis, a large percentage of renal dwarfs have marked dilatation and hypertrophy of the bladder with varying degrees of hydronephrosis and dilatation of the ureters. Fourteen cases out of seventeen examined showed these changes. This dilatation of the urinary tract cannot be explained, but it may be due to achalasia of the urethro-vesical sphincter. It can be demonstrated by pyelography, either retrograde or intravenous, and cystography. The differential diagnosis on radiological grounds between ordinary rickets and the rachitic type of renal rickets is difficult, if not impossible. The woolly type is pathognomonic, being peculiar to this disease. The only other condition remotely resembling it is congenital syphilis, where there is irregularity at the metaphysis.

However, the absence of periostitis, lack of symmetry, and the subperiosteal defects in the long bones should decide the cause. Failing these signs, the skull should be X-rayed: if it is involved, the diagnosis is easy.



FIG. 465.—Renal dwarfism. The woolly type in a boy of 15 years.

## GIANTISM AND PRECOCITY

The growth hormone of the pituitary, the gonadotrophic hormone, and the sex hormones may be concerned both in precocity and in giantism. Two effects may result: firstly, premature union of epiphyses, and secondly, giantism. When the lesion is in the adrenals, gonads, or pineal, sexual and mental

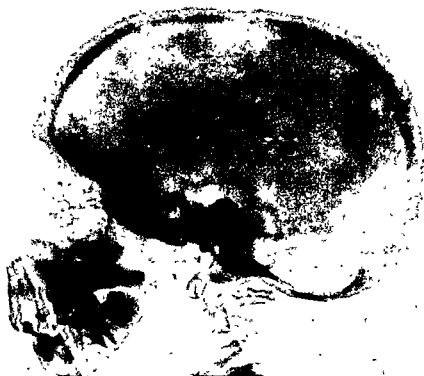


FIG. 466.—Renal dwarfism. The skull of the same patient as in Fig. 465, showing changes resembling those of *ostetis deformans*.

precocity appear and there is premature development of secondary sex characters. The genitals are usually large. Adrenal tumours tend to accentuate male characteristics, so that girl children develop facial hair, the body conforms to the male shape, and the clitoris enlarges. Pineal tumours usually affect boys, while tumours of the gonads are common in girls.

RADIOLOGICALLY, the ossification is seen to be advanced and the epiphyses appear and unite early. For this reason giantism is not seen in these cases. *Le Marquand* and *Russell* report a case of *pubertas præcox* in a boy associated with a tumour in the floor of the 3rd ventricle. We have a similar case showing a calcified mass in this region. The epiphyses have appeared very

early and are uniting with the shaft many years in advance of the recognised age (Fig. 467)

The so called eunuchoid giant shows a delay in union of the epiphyses following their late appearance. The subject is abnormally tall, slender, and of poor physique. The genitals are infantile and secondary sex characters either do not develop, or develop very late. The gonadotrophic hormone is



FIG. 467. Irioccity in a boy age 13 years. The size and development of the bones compared with those of a normal control of the same age and sex are excessive. There was a colloid tumour in the region of the floor of the third ventricle of the brain.

probably at fault. If the endocrine changes occur before union of the epiphyses of the long bones, gigantism will result. In certain cases no delay of ossification nor epiphyseal union can be found in the skeleton.

### ACROMEGALY

This condition is due to overactivity of the eosinophil cells of the anterior lobe of the pituitary and arises in adult life when all bone growth has

ceased and the epiphyses are united. In spite of this bone enlargement takes place in the hands, feet, and parts of the skull. It is possible that other bones are affected also, but the degree is too slight to be detected. All the viscera are enlarged, the tongue, lips, nose, and ears especially so. Sexual function is reduced or absent.

**IN THE SKULL** the sella turcica is enlarged. The degree of enlargement varies from being so slight as to escape notice to total destruction of the dorsum sellæ. The enlargement is almost entirely in the gland, so that a general expansion of the fossa is produced. The sphenoidal air cells may be encroached on until they are completely obliterated. The whole vault of the skull may be thickened and the accessory sinuses, especially the frontal, enlarged. The mandible is often increased in size and becomes prognathous. As a result of these changes the face becomes entirely altered. The supra-orbital ridges are so prominent that the forehead slopes backwards, the nose as well as the lower jaw becomes large and prominent. The malar bones are enlarged.

**IN THE TRUNK** it is common to find marked kyphosis, which adds to the general grotesque appearance and makes the arms appear unusually long and to hang from the shoulders in an ape-like manner. In all bones muscular attachments are accentuated and small bony outgrowths form at their insertions. The antero-posterior diameter of the chest is increased.

**IN THE HANDS AND FEET** in addition to the general increase in the size of the bones and the accentuation of the muscle attachments, the terminal phalanges become expanded into tufts which consist largely of cancellous bone.

### BASOPHILISM

The pluriglandular syndrome hitherto supposed to be of adrenal cortical origin is now known as basophilism because it is constantly associated with a loss of granules in the basophil cells of the pituitary and sometimes with an adenoma of these cells. The discovery that hormones originating in the pituitary control other endocrine glands in the body affords in part an explanation of basophilism. The syndrome appears to be commoner in women than in men, although this is possibly due to the unusual combination of amenorrhœa, adiposity, and hetero sexual hirsuties which arrest attention. The cases show a rapidly acquired, peculiarly disposed and usually painful adiposity confined to the face, neck, and trunk. There is a dusky or plethoric appearance of the skin with purple striae especially on the abdomen, together with a tendency to hypertrichosis of the face and trunk in the female and in the pre-adolescent male. This is accompanied by a sexual dystrophy shown by early amenorrhœa in the female and impotence in the male. There is persistent hypertension associated in advanced cases with retinitis and albuminuria. Glycosuria, hyperglycæmia, and sometimes erythræmia have been recorded. Backache, abdominal pain, undue fatigue, and extreme weakness may occur. Osteoporosis

is sometimes seen, and the patient may then have spontaneous fractures and become round shouldered, even with a measurable loss of height

This syndrome can be differentiated from that associated with a primary tumour of the adrenal cortex only by estimation of the urinary androgens. Virilism is to be ascribed primarily to cortical adrenal tumours only when serial sections of the anterior pituitary at autopsy fail to reveal either a basophil adenoma or loss of the granules in basophil cells. It is difficult to believe that the obesity of pituitary basophilism is due to pressure upon neighbouring cerebral tissues—it is almost certainly due to secondary involvement of other endocrine glands.

Radiologically, the only feature is lack of calcium in the bones. This is general and not confined to the bones of the trunk. It is more noticeable in the trunk owing to the fact that the soft tissues are enormously increased in this part of the body, and as a result the shading of the bone is less clear. The osteoporosis may be slight, but in extreme cases bones may bend and crack, causing marked deformity. This is best seen in the ribs. In the skull it is unusual to see changes other than lack of calcium. It is rare to find any alteration in the size of the sella turcica.

Neoplasm of the suprarenal body may arise in the cortex or in the medulla.

(i) *Neoplasm of the Suprarenal Cortex*—The so called carcinoma of the suprarenal cortex causes a sequence of signs and symptoms indistinguishable from Cushing's basophilism. There may be no detectable abdominal lump, and the correct diagnosis may be made only after perirenal air insufflation or even by surgical exploration. At times the tumour may grow to a very large size and practically fill the abdomen.

(ii) *Neoplasm of the Suprarenal Medulla*—Neuroblastoma of the suprarenal medulla appears clinically in two forms. The "Pepper" type is of no radiological interest. It is usually seen in infants, metastases spread rapidly to essential organs, and death occurs early. The "Hutchinson" type affects young children, usually below the age of 10 years. The abdominal mass is usually small and escapes notice until bone metastases have been present for some time. Metastases are common in the skull, especially in the region of the orbits. Irregular masses in the pericranium and exophthalmos are often the first signs. Radiologically, the appearance in the skull is indistinguishable from sarcoma and shows fine spicules of bone radiating from the outer table. In the long bones, however, the periosteum may be raised throughout the length of the shaft by a sheath of subperiosteal growth, which does not alter the outline of the cortex until very late in the disease. This appearance may be diagnostic. The differential diagnosis between this disease and congenital syphilis, chronic leukaemia, and scurvy should not be difficult.

## CHAPTER XLI

### BONE AND JOINT CHANGES IN TOXIC CONDITIONS

#### LEAD POISONING

THE METABOLISM of lead in the body is similar to that of calcium. Factors favouring the storage of calcium in the bones will also aid the deposition of lead. Tertiary lead phosphate stored in the skeleton is relatively harmless. In the bones of adults it cannot be detected radiologically, but in children

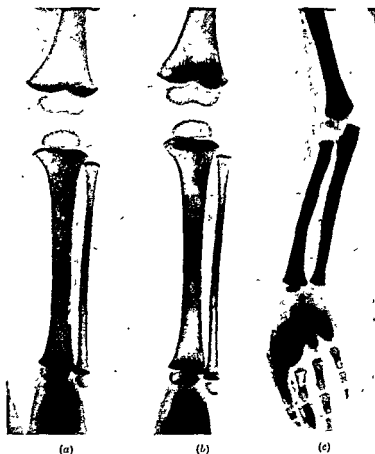


FIG. 468.—Lead poisoning in a child aged 2½ years. The deposits of lead salts are seen at the growing ends of the bones. (a) and (c) were taken when the child was first seen, but (b) was taken three months later, during which time no lead had been swallowed and so apposition of bone of normal density had occurred to the epiphyseal side of the lead line.

lead phosphate is deposited in the growing ends of bones, together with calcium phosphate, and it can be detected in radiograms. White lead used in face powders has been a source of poisoning among nurslings in Japan for over two centuries. In Queensland, Australia, lead used in painting verandah railings is readily powdered by the hot dry atmosphere, and gives rise to many cases of poisoning in children. In the United States lead paint from furniture, woodwork, or toys has been the commonest source of poisoning in children. In many of these cases perverted appetite, whether from mental deficiency, ill-health, or habit, has been noted. Water from lead pipes, lead foil, lead ointments, lotions, plasters, dusting powders, and lead nipple shields are other less frequent sources.

**Radiological Features.**—The wrists and ankles are the most satisfactory joints to examine, as here the growth is greatest. The lead appears as a perfectly clean-cut band of greatly increased density at the diaphyseal end of the growing bone (Fig. 468). There is no irregularity and no raising of the periosteum. The epiphyses are unaltered. The degree of density of the lead line depends on the rate of absorption of the metal, and its width on the length of time the absorption has been taking place. If the ingestion of lead is stopped, the deposition of the lead salts ceases and bone of normal density and texture is deposited on the epiphyseal side of the lead line.

Poisoning by *bismuth*, *strontium*, *phosphorus*, *fluorine*, and *vitamin D* may give rise to somewhat similar appearances. In such cases the exact nature of the poisoning must be determined by clinical and laboratory methods. The absence of irregularity and periosteal changes should prevent confusion with the less-dense band seen in congenital syphilis. In scurvy the ground-glass appearance of the shaft and epiphysis and the zone of increased translucence behind the dense band, together with the possible presence of subperiosteal hæmorrhages, make the diagnosis clear. The condition could scarcely cause confusion with healing rickets. The marble bone disease of Albers-Schönberg offers the greatest difficulty, and may be distinguishable only by clinical methods. This disease is discussed fully elsewhere.

### KASHIN-BEK'S DISEASE

This disease is endemic in Transbaikalia, Siberia, and is believed to arise from drinking water contaminated by manure. It may be a deficiency disease. Deformity of the hands; with shortening and rarefaction of the long bones swelling of the joints, and atrophy of muscles are the outstanding features.

### HYPERTROPHIC OSTEOARTHROPATHY

In this condition new bone is formed subperiosteally in the bones of the limbs; it is associated with certain diseases of the lungs or pleura. It is most commonly found in conjunction with chronic lung abscess, bronchiectasis, and carcinoma of the lung or bronchus.

There is always clubbing of the fingers and toes and sometimes swelling of the joints of the hands feet wrists and knees. In advanced case thickening can be felt at the ends of the long bones and there may be pain in the areas where the process is most advanced.

In most of the cases seen by the authors chronic pulmonary disease was present but it has been reported that osteoarthropathy can be present with any chronic infective process with heart disease, and also with Hanot's cirrhosis of the liver. We have histological proof of its presence in a case of polyposis of the colon.

*Morbid Anatomy*—The condition is a chronic periostitis with abundant new bone formation. Histological sections show superimposed on the old cortex, close meshed new spongy bone formed by the periosteum. The old cortex may show osteoporosis.

Radiologically, the condition is best seen at the wrist and elbows. It is usually stated that the metacarpals and phalanges are the most satisfactory sites to examine, but this is not so. The metacarpals tend to be misleading especially in advanced cases where the subperiosteal bone deposits are well established, so that they appear to be accentuated muscle attachments on thickened shafts.

The subperiosteal deposits are not confined to the ends of the shafts of the bones but run along the whole length. The periosteum is raised unevenly so that the outline appears serrated and the deposits beneath it are unevenly calcified or ossified giving a lace work effect. In long standing cases bone is definitely formed and this is of uniform density (Fig 469).



FIG 469—Hypertrophic osteoarthropathy. The uneven raising of the periosteum at the bones of the forearm in a woman with chronic lung infection with abscess formation for four years.

### PSORIASIS ARTHROPATHICA

Menzen states that the condition of pain in the joints associated with psoriasis was first described by Alibert in 1882, Bain in 1860 applied the name psoriasis arthritica to it. It is a polyarticular condition which may attack any joint. The joint pains are usually prominent during an exacerbation of the skin condition and tend to recover as the skin improves. During an attack the joint becomes painful and swollen and in the acute stage the bones of the joint may be separated by an effusion. After repeated attacks the cartilage becomes eroded with subsequent loss of joint space, the joint surfaces may be uneven.



## CHAPTER XLII

### BLOOD DISEASES WITH ASSOCIATED BONE CHANGES

#### ERYTHROBLASTIC ANÆMIA OF COOLEY

THIS CONDITION, described by *Cooley* in 1927, is a familial disease which he claims is peculiar to children of Mediterranean stock. It has been well known in Greece since 1910, but it is very rare in England. The children are mongoloid in appearance, owing to anæmia and thickening of the bones of the skull. The abdomen is prominent as a result of enlargement of the liver and spleen.



FIG. 47. Erythroblastic Anæmia (Cooley's case).

and the anaemia is marked. Large numbers of erythroblasts are seen in blood films. The outcome is always fatal.

Radiographically the bones show a lack of calcium with accentuation of the bone pattern. The cortex is thin and *Logt* and *Diamond* state that the marrow may invade it causing punched-out areas. The appearance may be indistinguishable from osteitis fibrosa. The skull shows great thickening which takes place between the inner and outer table, the diploe being prominent. In the later stages the surface of the skull becomes covered with radiating spicules (Fig. 4.0).



Control

FIG. 4.1.—Leuco-erythroblastic anaemia in a woman aged 61. There is slight increase in density of the bone when compared with that of a normal control.

#### SICKLE CELL ANÆMIA

This disease is familial and confined to people of negroid descent. The name is derived from the sickle-shaped erythrocytes circulating in the blood. Progress is accompanied by exacerbations and remissions. Haemolysis is accompanied by jaundice, marked during the exacerbations. Radiographically

the bone changes are similar to those seen in Cooley's anaemia but are considerably less marked.

#### FAMILIAL HÆMOLYTIC ICTERUS

Increased fragility of the erythrocytes with hæmolytic icterus anaemia and splenomegaly are seen. Bone changes are usually absent or slight as in sickle cell anaemia.

#### LEUCO ERYTHROBLASTIC (OSTEOSCLEROTIC) ANÆMIA

Conditions such as carcinomatosis and myelomatosis which may give rise to leuco erythroblastic (myelophthisic) anaemia are considered elsewhere. Occasionally this anaemia occurs apart from either of these diseases. It

is very chronic and shows enlargement of the spleen and changes in the bones. It divides the osteosclerotic diseases into two types those with myelosclerosis and those with myelosclerosis. The term osteosclerotic is sometimes used but the name given by *Turnbull* leuco erythrocytosis is the most satisfactory. *Lauglan* has collected seven



Fig. 47. Myeloid leukemia. Deposits in the skull of a patient aged 41 years, who was known to have myeloid leukemia for two years.

cases of myelosclerosis which have appeared under a variety of names as aleukæmic myelosis with osteosclerosis. The etiology is unknown. Radiologically there is an increase of connective tissue or bone in the medullary cavity while the cortex remains unaltered. There is no tendency to fracture.

Radiologically the medullary cavity shows an increased density while the cortex still remains recognisable. The increase of density may be very slight and controlled radiograms must be taken. If the pathological process is one



spleen are prominently involved. The whole of the alimentary tract and the bone marrow contains lymphadenoid tissue which also may become involved. Other diseases, notably tuberculous adenitis and lymphosarcoma, may in some respects resemble Hodgkin's disease, and in many cases the diagnosis can only be made by means of the microscope, a portion of tissue being removed for biopsy examination. It is perhaps insufficiently appreciated that in 10 per cent of cases of lymphogranuloma the skeleton becomes involved. *Carrier* and *Copeland* estimate the number at 15.7 per cent and quote *Zeigler* as putting it as high as 40 per cent.

The involvement of bones usually occurs late in the disease, although *Dresser* and *Spencer* mention two cases in which the clavicle was invaded, although there was no other manifestation of the disease in the body. Pain is the outstanding clinical feature and may exist for some long period before definite changes are detected radiologically. The pain may be the result of destruction of the bone or of pressure by a mass of lymphadenoid tissue on nerve roots. It may be accompanied by herpes zoster. The bone may be the site of lymphogranulomatous deposits, which are carried by the blood in the same way as malignant metastases are disseminated, or an adjacent mass of glands may become involved and spread in the bone by pressure and erosion.

The commonest sites in which to find bone deposits are the pelvis, adjacent lumbar spine and upper ends of the femora; any bone may be affected, and, if the spread has been by the bloodstream the deposits are usually multiple.

The radiological appearance, as well as the method of spread, resembles malignant disease. The commonest finding is irregular areas of destruction with little or no periosteal reaction; in the pelvis this may resemble multiple myelomatosis. At times definite reaction may occur and this may take the form of sclerosis or periosteal proliferation. When there is periosteal reaction the condition may resemble osteomyelitis or a primary malignant bone tumour. When sclerosis is marked, the appearance may resemble that of carcinomatous metastases of the osteoplastic type or of osteitis deformans. *Dresser* and *Spencer* show a case with a typical "ivory vertebra," such as is seen in malignant disease and osteitis deformans.

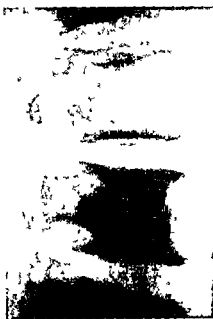


FIG. 474.—Hodgkin's disease involving the anterior aspect of the third lumbar vertebra.

In the spine destruction is usually the chief feature (Fig 474) and the bodies involved soon collapse causing deformity and often paralysis. Involvement of the abdominal glands may result in spread to the lumbar vertebrae with consequent erosion of the anterior surface of the vertebral bodies. The intervertebral discs escape and since the same appearance is also seen when aneurysm erodes the bodies the two conditions may be indistinguishable radiologically unless the walls of the aneurysm are calcified.

In the long bones there may be destruction and periosteal proliferation similar to that seen in osteoclastoma. Fractures may occur.

Usually the diagnosis is easy as the disease has been established and recognised for some time before pain draws attention to it. Spread to the bones

## CHAPTER XLIII

### CARCINOMATOSIS AND MULTIPLE MYELOMATOSIS OF BONES

#### CARCINOMATOSIS

SECONDARY CARCINOMATOUS deposits in bones may stimulate either osteoclastic resorption or osteoblastic apposition or both. Usually resorption preponderates and a condition of osteoporosis with a tendency to fracture



FIG. 47a. Osteoclastic secondary carcinoma in a woman who had the right breast amputated for carcinoma three years previously.

results. It is known as osteoclastic carcinomatosis. If apposition preponderates, the resulting condition is osteoplastic carcinomatosis. The two conditions can readily be distinguished in radiograms. Mixed forms are known to occur.

In 1935 the calcium balance in ten cases of carcinomatosis of bones was studied. Where the changes are mainly osteoclastic the calcium output may be two or three times the normal. In osteoplastic carcinomatosis the calcium balance is actually positive—that is to say the patient excretes less calcium than is present in the diet. Under these conditions the serum calcium figure may be 8.5 or 9 mg per 100 c.c. In all other circumstances the serum calcium and plasma phosphorus figures are normal in carcinomatosis of bone. The plasma phosphatase is always raised.

Primary carcinoma of the prostate commonly gives rise to secondary osteoplastic carcinomatosis and at times malignant tumours of the thyroid



FIG. 4.6. Osteoplastic secondary carcinoma in a man with primary carcinoma of the prostate.

ovary and stomach have the same effect but there is no hard and fast rule. Carcinomatosis is much less frequently encountered than solitary discrete secondary deposits.

**RADIOGRAPHICALLY** osteoclastic carcinomatosis is seen in two forms. In the first the condition is manifested by lack of calcium and therefore may be very misleading and indistinguishable from many other generalised diseases affecting bone. The bones gradually become less dense the patient usually dying from asthenia before the shafts of the long bones break or bend.

In the second type in addition to the general lack of calcium almost every bone in the body is peppered with areas of translucence varying in diameter from 1 mm to 3 cm. These areas may become confluent causing



fractures and bending of the bone. The areas are irregular in outline. They rarely occur below the elbows and knees and therefore may be distinguished from multiple myelomatosis (Fig. 475).

In the *osteoplastic* type in the early stages small discrete areas of opacity are seen scattered over the bones of the trunk and limbs. They are more likely to invade the bones below the elbows and knees at an early date than the osteoclastic variety; they spread throughout the whole bone instead of being confined to the ends as in osteoporosis. As the disease progresses these areas spread and become confluent so that the whole bone becomes dense (Fig. 476). At this stage it may closely resemble the amorphous type of osteitis deformans though of course the bones show no widening.

Discrete secondary carcinomatous deposits are described in the section on tumours of bones.

THE DIFFERENTIAL DIAGNOSIS may be difficult. When the only feature is lack of calcium the diagnosis must be sought from the history, the clinical evidence and the blood chemistry. The osteoclastic type is likely to be mistaken for myelomatosis but the irregular shape of the invaded areas and the absence of the clean cut appearance seen in myelomatosis will decide this; it can sometimes be confirmed by demonstrating the presence of Bence Jones protein in the urine. In most cases the primary growth will be evident.

### MULTIPLE MYELOMATOSIS

For obvious reasons the multiple marrow tumours in this disease may lead to its confusion with a generalised disease of bone. It arises in the blood forming cells of the bone marrow and therefore first appears in the skull, spine and ribs. Later extensions of red marrow may occur in the long bones and then the lesions of myelomatosis may be found in all the bones of the skeleton. Examination of the urine shows in almost every case the Bence Jones protein.

The whole skeleton should be examined. The tumours are characteristic appearing in the radiograms as clean cut elliptical or circular areas of complete translucence varying from 1 mm. to 3 cm. in diameter. The surrounding bone shows lack of density. There is no attempt at local reaction either in the bone itself or in the adjacent periosteum. Fractures and areas of collapse are common especially in the vertebrae. In most cases particularly late in the disease the long bones are affected.

In typical cases the diagnosis is easy—carcinomatosis is the principal difficulty. As a rule in secondary carcinoma the areas have a woolly and irregular outline whereas those of myelomatosis are often quite clean cut. Secondary carcinomatous deposits are less often found below the elbows and knees than are the lesions of myelomatosis. The circular deficiencies of the skull may resemble those seen in osteomalacia and generalised osteitis fibrosa. In such cases the other bones will help to make the diagnosis clear and the chemistry of the blood and of the urine must be considered.

## CHAPTER XLIV

### DISEASES OF UNKNOWN ORIGIN

#### LEONTIASIS OSSEA

When *Virchow* suggested the use of the term *leontiasis ossea* in cases of hyperostosis of the skull he had in mind *fibroma molluscum* in which masses of new connective tissue develop in the skin. He believed that the over growth of bone in hyperostosis corresponded exactly to elephantiasis of the soft parts and he decided to call these cases *leontiasis ossea* not because the bone disease produced a leonine appearance but because he considered it to be analogous to a disease of the soft parts which did.

To-day the term *leontiasis ossea* is used symptomatically and not in a specific sense. It includes cases of *osteitis fibrosa*, *osteitis deformans* and chronic *periostitis*. Those cases due to *osteitis fibrosa* include both the general diffuse osteitis of the cranial and facial bones and osteitis beginning in one or both jaws and rarely spreading far beyond. A case has been described where a man aged 34 had had a gradually increasing swelling of the mandible for fifteen years until it measured 8 by 6 by 4 inches. Rarely in *Paget's disease* the jaws may be so much involved as to stand out in contrast to the other bones. Finally there are cases of a very chronic *periostitis* spreading slowly from bone to bone to which the title of *creeping periostitis* of the bones of the face and skull may be given. This type begins in almost all cases in the nasal fossæ or sinuses. In early cases the fossæ may be blocked with bone and later the involvement of other bones may cause a coalescence of the periosteal thickening which has been likened to approaching streams of osseous lava. Complications include obliteration of the nasal duct, *exophthalmos* and optic atrophy.

#### OSTEOPETROSIS

*Osteosclerosis fragilis generalisata*, *marble bones* and *Albers Schonberg's disease* are all synonyms for this disease. It is very rare and though originally described by *Albers Schonberg* in 1904 it was not until 1914 that another case was reported. The name *Albers Schonberg's disease* is unfortunate as in 1915 the same author described *osteopetkilosis* for the first time and this is sometimes referred to as *Albers Schonberg's disease*.

The disease appears to be congenital and may be familial. The age at which the condition is seen varies—*Albers Schonberg's case* was 26. In 1930

*Pirie* showed changes in the bones of a fetus whose mother showed well developed osteopetrosis, and whose development he followed for six years

One of the commonest symptoms is fracture of a limb. In advanced cases a severe anæmia may appear, and the liver, spleen, and lymph glands may be enlarged

**Radiological Features**—At present the diagnosis of osteopetrosis is a purely radiological one. The outstanding feature is a homogeneous density of the bones and this appears to be due not to the spread of the cortex at the



FIG. 477.—Osteopetrosis in a boy aged 17 years admitted with a fracture of the neck of the femur following slight trauma. The bones are unaltered in size. (Dr. Rolan Williams case.)

expense of the cancellous bone, but to the laying down of some material of high calcium content in the medullary cavity. This material is relatively soft, though its calcium content makes it give a dense shadow. Thus a bone which looks as if it should be ivory hard is really brittle (Fig. 477).

The condition starts at the ends of the diaphyses and travels towards the mid point of the shaft. In the early stages it appears as a dense band, very similar to that seen in lead poisoning (Fig. 478). Later the ends of the bones affected by the process, widen and become club shaped, while the density appears further up the shaft. All bone texture is lost in the affected region. *Pirie* reports a "ring shadow" about the carpal bones due to a circle

of dense bone. He also states that in one of his cases which came to post mortem the 'marble' bone cut like chalk with a knife.

All bones are not necessarily involved and the process does not affect the whole shaft until the age of puberty. In the skull the density is marked at the base so that all air spaces and foramina tend to be obliterated thus may bring about optic atrophy from pressure.

*Karschner* reports *hydrocephalus* in his infant cases and says all cases are physically under developed and have a peculiar facies. The teeth are always defective. The epiphyses are slow to appear and union with the shaft is delayed. *Brailsford* describes the expanded ends of the phalanges as having an appearance suggesting cracks vertical to the epiphyseal line. His case also showed the density to be in plaques of varying width parallel to the epiphyseal line.

IN DIFFERENTIAL DIAGNOSIS lead poisoning in a child may resemble early osteopetrosis. If the possibility of further inges-

tion of the poison is removed new bone of normal texture will form to the epiphyseal side of the line of density. The faeces and urine should be collected for a week and analysed for lead and the blood examined for punctate basophilia. In all cases of osteopetrosis a blood count should be done.

Congenital syphilis may be ruled out by the absence of periostitis and the



FIG. 478.—Same case as FIG. 477. The increased density is seen to be at the upper and lower margins of the vertebral bodies.

negative Wassermann reaction. Confusion with osteitis deformans of the amorphous type and secondary carcinoma is not likely to arise. Since so little is known of the pathology of this condition it is the more important that controlled radiograms should be taken in all cases.

### MELORHEOSTOSIS (LERI)

It is impossible to ignore this condition while discussing osteopetrosis. Melorheostosis was first described by Leri and Jonamy in 1922. It occurs in a limb, and when radiograms are taken deposits of dense bone are found running down the surface of the bones, causing thickening. The medullary cavity is also involved so that the bony thickening is not entirely extraosseous. Leri watched his case over six years and found that as time advanced other bones became involved though the patient did not suspect this. The condition resembles osteopetrosis in some respects but at present it is not known if the two are related.

### FOCAL OSTEITIS FIBROSA

This occurs much more commonly than the generalised disease. It is a condition affecting one or more bones, usually not disabling of slow progress, and showing a tendency to become arrested. It occurs chiefly in adolescence, and is often symptomless until spontaneous fracture occurs. The figures for serum calcium and plasma phosphorus are invariably normal—a finding in striking contrast to that of the generalised disease. The calcium balance is usually normal and taken in conjunction with the normal blood chemistry, this finding is of far reaching importance. It is evidence strongly against hyperparathyroidism and therefore although a number of bones may be affected the condition is totally different from the generalised disease and exploration of the neck is unjustifiable.

It is generally the ends of the shafts of the long bones which show changes in radiograms. The lower jaw is a common site. Often only one bone is involved. It is



FIG. 479.—Osteoclastoma in the humerus of a child. The surrounding bone is of normal density.

usual to find more than one third of the shaft affected. The uniform enlargement of the bone, containing cyst-like areas, usually divided by a few coarse trabecular strands, is seen. The cortex is thin and expanded by the cysts, the periosteum is normal. The adjacent bone is normally calcified (Fig 479). If a long bone breaks, union is usually strong and the cyst like area becomes filled with bone. The vault of the skull is unaffected, but occasionally a great increase in density and actual thickening of the bone of the floor of the anterior and middle fossæ may be seen. It is difficult to differentiate between this disease and osteitis deformans in some cases, and it may be necessary to follow the progress over a period of time before a definite conclusion is reached. Giant celled tumours are solitary, and show little trabeculation. They represent what is really a special form of focal osteitis fibrosa. They tend to expand the cortex to a greater degree, at times bursting through into the surrounding tissue.

### HÆMOPHILIA

Hæmophilia is a constitutional disease characterised by a life long tendency to prolonged hæmorrhage and a markedly delayed coagulation time. The nature of the disturbance on which the tendency to bleed depends is not understood. Hereditary transmission depends upon a sex linked recessive Mendelian characteristic. Hæmophilia is dominant in the male and recessive in the female. Women themselves never suffer from the disease, but transmit it to some of their male offspring.

The clinical manifestations include prolonged bleeding from cuts and other injuries, epistaxis, hæmorrhage after extraction of teeth, subcutaneous hæmatomata often large and spreading and hæmorrhages into the joints. These hæmarthroses occur mainly in the large joints especially the knees and elbows as these are prone to injury. The blood may be absorbed completely and leave no sequel or organisation and ankylosis may result.

A small single hæmorrhage cannot be recognised in a radiogram. It is usually only after repeated bleeding into a joint that demonstrable changes occur. In the acute stage, soon after a hæmorrhage the bones of the joint will be seen to be separated slightly and the distended capsule will limit an area of haziness, due to the effusion of blood. This is in no way distinguishable from any other effusion into the joint. After repeated hæmorrhages the appearance become indistinguishable from that of hypertrophic arthritis with diminution of joint space and unevenness of joint surface due to loss or destruction of cartilage. The margins of the joint surfaces will be sharpened and this may be a marked feature. The joint may be completely fixed.

In neither of the above instances is there any special feature by which the disease can be recognised. The chance to diagnose the condition by X ray rarely occurs in England since the family concerned usually knows of the presence of the bleeder strain and therefore supplies the diagnosis. However,

in a few cases an ankylosed joint shows changes which are peculiar to this disease alone. Cyst like areas are seen in the subchondral bone and may run from the joint surface into the shaft of the bone, sometimes to the depth of  $\frac{3}{4}$  inch, they are quite clean-cut and show no surrounding density, when they are numerous the end of the bone presents an appearance resembling a lattice (Fig 480). The joint space is much decreased and the joint surface is largely destroyed. Even if ankylosis has not taken place, there is usually considerable diminution of movement. Calcification or ossification arising in organised blood clot or thickened synovial membrane may be seen.

In explanation of these appearances Alason assumes that after repeated hemorrhages the clot irritates the synovium which becomes villous. The cartilage loses its gloss and is covered with fibrin and if this fibrin is removed defects are found in the cartilage, sometimes extending into the bone.

In the knee joints the intercondylar fossa may be much widened and deepened. It has been suggested that this is due to hemorrhage into the attachments of the crucial ligaments. The bones about the affected joint may show loss of density.

#### OSTEITIS DEFORMANS *syn* PAGET'S DISEASE

Osteitis deformans is not inflammatory in origin it seems likely that it is a disorder of mineral metabolism. Histologically, there is tremendous resorption of bone associated with tremendous apposition completely altering the normal architecture, but apposition predominates. The sexes are affected in the proportion of three men to two women. The disease rarely begins before the age of 40, and the commonest age of onset is 55. It may remain symptomless for ten years or more. It is very slow in progress, and rarely influences the general health, giving rise to few troubles apart from those which are due to changes in the shape of the bones. The tibia, femur, and sometimes the radius and ulna, become enlarged and bowed. The bowing usually takes place in such a manner as to accentuate the normal curve of the bone. After the condition has been present for many years the calvaria becomes thickened and kyphosis occurs. In its earlier stages, and sometimes throughout its course,



FIG 480.—Hemophilia. The lattice appearance is seen in the bones of the elbow joint after repeated hemorrhages into the joint.

the disease is attended by pains in the affected bones pains widely various in severity and not especially nocturnal or periodical The bones are affected in the following order of frequency pelvis spine femur tibia skull fibula clavicle humerus and radius The arteries are usually thickened and tortuous and the blood pressure often raised Occasionally a patient with osteitis deformans shows an abnormal tendency to fractures Osteogenic sarcoma may occur

Histological and chemical investigations have proved beyond doubt that generalised osteitis fibrosa is unrelated to osteitis deformans A differential diagnosis between the two conditions on radiological grounds alone is never justified Enlargement of the parathyroids has not been demonstrated in osteitis deformans and therefore exploration of the neck should never be undertaken The serum calcium and plasma phosphorus are normal Studies of the calcium balance have shown no departure of any magnitude from the normal The plasma phosphatase is constantly high as in many other generalised diseases of bone

Excessive resorption of bone leads to irregular osteoporosis and it is presumably this process which leads to deformity The new bone which replaces the old appears in radiograms in two forms which may be called the spongy and the amorphous The two types are often found in the same patient and it must be realised that the subdivision is one of convenience only Whether the appearances represent two different types of histological change is not known The spongy form is the more common It consists of coarse irregular striæ arranged either as parallel trabeculae or running in the direction of the normal lamellæ of a cancellous bone The amorphous form is a generalised opaque deposit producing a granular mortar like appearance Whether the two processes take place simultaneously whether the amorphous stage precedes the spongy stage or whether the spongy stage can appear without any preceding amorphous stage is not evident Where a bone is affected by the spongy changes its diameter is increased sometimes to a marked degree and in the medullary cavity the trabeculae are accentuated and too widely separated giving a streaky appearance The corticalis is partly or entirely replaced by bone similar to that seen in the medullary cavity and in an extreme case the impression is that the whole bone consists of cancellous tissue highly magnified The criss cross trabeculae which are seen in the central portion of the long bones are probably not medullary but are the cortical trabeculae seen broadside on through the more translucent medulla Indefinite cyst like areas of irregular shape may be seen scattered about the bones

Where the changes are of the amorphous type the normal bone texture is entirely replaced by chalky homogeneous shadows The diameter of such bones is usually increased

In the pelvis either spongy or amorphous type may be found though it is more usual to find a mixture of the two The whole pelvic girdle is



increased in size so that it is often impossible to include the pelvis on a 15 by 12 inch film, and it is necessary to use a 17 by 14 inch. The spongy type of change produces a larger pelvis than the amorphous type. In marked cases there may be very great deformity, and in almost all cases the pelvic brim is triangular and the outlet is decreased in size, partly owing to the

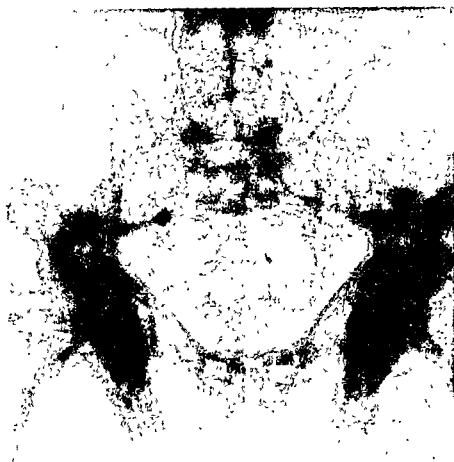


FIG. 481.—Osteitis deformans of the spongy type, showing widening of the bone with criss-cross trabeculation.

increase in size of the bones, and partly to the thrust of the femoral heads upward and inward on the softened bone. In most cases the iliac crests are thickened and splayed out, the iliac bones thus taking on a massive appearance. In the sacrum coarse trabeculae run out over the sacro-iliac joints, giving the impression of thick calcified threads binding the sacrum to the iliac bones (Fig. 481). Part or the whole of the pelvis may be involved, and some areas may be amorphous and others spongy. Irregular cyst-like areas are common. In

the amorphous type not only is the bone often widened but it may contain cyst like areas. This fact is important in differential diagnosis as examination of the spine alone may be very misleading the amorphous type of osteitis deformans being indistinguishable from secondary carcinomatosis of the osteoplastic type (Fig. 482)

In the spine both spongy and amorphous types are found. In the former the vertebrae involved give the impression of having been crushed. The bodies are wider and flatter so that the upper and lower surfaces are nearer to each other than normal. The whole bone pattern is accentuated and coarsened.



FIG. 482. Osteitis deformans of the amorphous type

and the detail is not clear. In the amorphous type the bodies are less likely to be flattened and the condition may therefore be mistaken for that of secondary carcinomatosis of the osteoplastic type. At times one or two vertebrae alone may be involved. The solitary *vertebra nigra* is therefore not proof of secondary carcinomatous deposits. Marked kyphosis is present in advanced cases. This together with the increase in width of the bone may cause encroachment on the spinal canal resulting in compression paraplegia. Angioma of a vertebral body could possibly be mistaken for osteitis deformans of the spongy type but fortunately it is very unusual to find a single vertebra affected with no evidence of changes in other bones in osteitis deformans.

Changes in the skull begin chiefly in the outer table. Irregular calcification results in the appearance of islands of dense bone in parts of the skull seen broadside on and gives the appearance of irregular excrescences and indentations of the parts seen tangentially. As uneven thickening of the outer table progresses the differentiation between outer and inner tables becomes lost, and finally the inner table also loses its clarity of definition and shows the same ill defined appearance as the rest of the bone. In many cases the sutures are seen to be obliterated (Fig 483). In the later stages the thickness of the calvaria has become increased to several times that of the normal. The bone then appears to be made up of numerous ragged patches and tufts of calcified

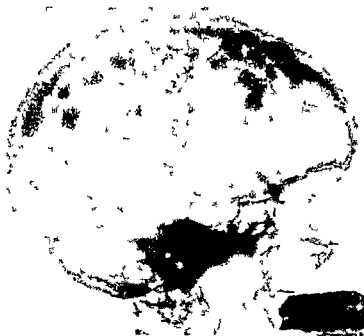


FIG 483 Osteitis deformans showing uneven thickening of the outer table of the skull

tissue with more translucent areas between them. In cases where the skull alone is considered the appearances may be similar to those seen in renal rickets but no confusion is possible owing to the different age incidence. At times an area of definitely circumscribed osteoporosis (osteoporosis, circumscripta) is seen in the skull, it is most commonly found in the frontal region and usually involves the greater part of the frontal bone. It is a manifestation of osteitis deformans, and when other bones are examined signs of the spongy type of the disease are found (Fig 485).

In the bones of the limbs, combinations of amorphous and spongy types are usually found. There is increase in thickness of the bone, the cortex often becoming several times its normal thickness. It usually shows coarse irregular

striae running mainly in a longitudinal direction. It is common to find the bones bent especially the tibia, femur, radius and ulna. At the points of maximum bending cracks which are really incomplete subperiosteal fractures are often seen on the convex surface. As many as twenty may be seen in the anterior aspect of the tibia. They occur always in bone showing the amorphous type of change. On the concave surface of a bone which is bowed a peculiar folded or crumpled appearance is sometimes seen.



FIG. 484.—Osteitis deformans of focal distribution. No other bone affected.

Changes are sometimes seen in the tarsus but the carpus escapes. The os calcis shows coarse spongy trabeculation. The metatarsals, metacarpals and phalanges may be altered, an increase in density of amorphous type being the usual change. Rarely these small bones show small cyst-like areas. There is no apparent symmetry about the changes in the skeleton and one is struck by the apparently haphazard manner in which the bones of any particular patient may be affected. Thus on one side one rib may be involved together with another rib higher up or lower down on the other side or the metacarpal bone of one finger may be involved with the phalanx of another finger.

In a few cases the disease is apparently confined to one bone or to part of one bone—tibia, femur, humerus or clavicle. Where part of one bone is affected there is a definite line of demarcation where the abnormal ends and the normal begins (Fig. 484). Thus there may be very definite changes in the upper two thirds of the tibia while the lower third is normal. Sometimes part or one half of the pelvis is affected while the rest of the skeleton escapes. The local disease is always of the spongy type; it is often confused with focal osteitis fibrosa cystica.

Radiograms reveal the shadows of arterial calcification in more than 40 per cent of cases. Such calcified arteries are best seen in the lower limbs. Though it is true that osteitis deformans is chiefly seen in the elderly

such a high percentage suggests that a special cause operates. It is possible that the excess of phosphoric esterase in the blood accelerates and intensifies the deposition of calcium salts in degenerating vessels. There is no evidence of a higher incidence of renal or vesical calculus in osteitis deformans than in the normal.

Occasionally a patient with osteitis deformans shows an abnormal tendency to fractures. When a fracture occurs it may be in a situation and of a type not commonly met with in normal persons. Whenever a bone affected by osteitis deformans is radiographed after a recent fracture it is always in the

stage of amorphous calcification or of early fluffy trabeculation. In no case does a fracture take place when the bone is in the final well trabeculated stage.

Osteogenic sarcoma may occur in osteitis deformans. In one series of seventy-one cases of osteogenic sarcoma, osteitis deformans was found in 28 per cent. In all of these the osteitis deformans had been present from ten to fifteen years prior to the development of the complicating sarcoma. In radiograms the point of origin is usually seen to be subperiosteal and a mass is found situated chiefly in the soft tissues but attached to the bone. The evidence of malignant invasion of the bone is very small compared with the size of the



FIG. 485.—Osteitis deformans showing osteoporosis circumscribed. The pelvis and other bones show the changes typical of osteitis deformans of the spongy type.

mass. Osteoarthritis of the hip, knee and/or spine is sometimes found in long-standing cases.

### ATROPHIC CHANGES IN DIGITS

Certain conditions lead to inefficient blood supply to the extremities and in extreme cases this may lead to osteoporosis and resorption of bone especially in the terminal phalanges of the fingers and toes. The condition may be due to spasm of the arteries as in Raynaud's disease or to obliteration of the lumen of the vessel as in thromboangiitis obliterans. The bone either becomes absorbed gradually from the tip towards the base or becomes thinned

at the narrow wasted middle of the shaft of the phalanx until eventually the tip of the bone becomes separated. Examples of this are seen in Raynaud's disease and scleroderma thromboangitis obliterans (Buerger's disease) syringomyelia (Morvan's disease) diabetic gangrene, and leprosy. Thinning and narrowing of the bones may be seen in acrosclerosis. Other rare conditions giving the same appearance are frost bite ergot poisoning lupus mutilans sarcoid and Kaposi's hæmorrhagic sarcoma.

## BIBLIOGRAPHY

## General

- EWING JAMES Neoplastic Diseases 1934 Philadelphia W B Saunders Co  
 GESCHICKTER C F and COPELAND M M Tumors of Bone 1936 Amer Journ  
 Cancer New York  
 HARRIS H A Bone Growth in Health and Disease 1933 London Oxford  
 University Press  
 KNAGGS LAWFORD R Inflammatory and Toxic Diseases of Bone 1926 London  
 Simpkin Marshall Hamilton Kent & Co Ltd  
 KOHLER A Röntgenology 1935 London Bailliere Tindall & Cox  
 SCHINZ H K BAENSCH W and FRIEDL F Lehrbuch der Röntgendiagnostik  
 1939 Leipzig Thieme

## Achondroplasia

- HARRIS H A Bone Growth in Health and Disease 1933 London Oxford  
 University Press  
 KNAGGS LAWFORD R Achondroplasia (Chondrodystrophia Fetalis) Brit Journ  
 Surg 1927 28 XI 10  
 LEWIN P and JENKINSON E L Chondrogenesis Imperfecta Achondroplasia  
 Chondrodystrophia Fetalis Amer Journ Roentgenol 1924 XI 150  
 RISCHBIETH H and BARRINGTON A Treasury of Human Inheritance Eng Lab  
 mem 1912 XI Paris VII and VIII London Dulau & Co

## Osteopoikilosis and Voorhoeve's Disease

- ALBERS SCHONBERG H Skelettanomalie von Atavistischen Interesse Proc Supra  
 condyloideus oder Entepicondyloideus Fort a d Geb d Röntgenstrahlen 1915  
 XXIII 175 Fine Seltene bisher nicht beobachtete Strukturanomalie des  
 Skelettes Fort a d Geb d Röntgenstrahlen 1915-16 XXIII 174  
 BENTON P G K Roentgenological and Experimental Studies on the Pathogenesis  
 of the Dyschondroplasias Acta Radiol 1924 III 89  
 LEDOUX LEBARD R L'Osteopoikilie forme nouvelle d'ostéite Condensante  
 Généralisée sans Symptômes cliniques Journ de Rhum et d'Arthritisme 1936 II 133  
 PETERSEN G F Case of Osteopoikilosis Acta Radiol 1936 XVII 388  
 VOORHOEVE N L Image Radiologique non Encore décrite d'une Anomalie du  
 squelette Acta Radiol 1924 III 407

## Osteochondrodystrophy and Gargoylism

- ASHBY W R STEWART R M and WATKIN J H Chondroosteo dystrophy of the  
 Hurler Type (Gargoylism) A Pathological Study Brit 1937 LX 149  
 BINSWANGER E and ULLRICH O Über die Dysostosis multiplex (Typus Hurler)  
 und ihre Beziehungen zu anderen Konstitutionsanomalien Z Klin Med 1933  
 LIV 699

- RAILSTON, J. I., "Chondro osteo dystrophy; Roentgenographic and Clinical Features of Child with Dislocation of Vertebra," *Amer J. Surg*, 1929, VII, 404
- LY, R. W. B., SHELTON, W., and CARON, N. B., "Gargoylism," *Quart J Med*, 1936, V, 119
- FLMHOZ, H. F., and HARRINGTON, F. R., "A Syndrome Characterised by Congenital Clouding of the Cornea and by other Anomalies," *Amer J Dis Child*, 1931, XLI, 793
- UTLER, G., "Über einen Typ multipler Abartungen, vorwiegend am Skelettsystem," *Z Kinderheilk*, 1919, XXIV, 220
- LANGF, C., and WOLTING, L., "Multiple Skelettartungen, Hepatosplenomegalie, geistig-Rückständigkeit," *Acta paediatr*, 1936, XIX, 71
- BOQUJO, L., "Sur Une Forme de Dystrophie Osseuse Familiale," *Arch méd Enf*, 1929, XXXII, 129
- THILL, C. R., "Juvenile Amautrotic Idiocy," *Arch Neurol Psychiat*, 1934, XXXII, 198

### Osteogenesis Imperfecta

- FLI, J., "Blue Sclerotics and Fragility of Bone," *Treasury of Human Inheritance*, 1928, II, 269
- ROMFR, R. S., "Osteogenesis Imperfecta," *Amer. Journ Roentgenol*, 1933, XXX, 631
- RECH, G. E., and SODEMAN, W. A., "Hereditary Hypoplasia of the Mesenchyme," *Amer Journ Med Sci*, 1937, CXCV, 844
- ILLS, R. G., and McLANAHAN, S., "Brittle Bones and Blue Scleras in Five Generations," *Arch Int Med*, 1937, LXV, 41
- UNTER, D., "Case of Osteogenesis Imperfecta," *Lancet*, 1927, I, 9
- OHANSSON, S., "Ein Fall von Osteogenesis Imperfecta mit Verbreiteten Gefassverhalungen," *Acta Radiol*, 1921-22, I, 17
- NAGLE, LAWYARD R., "Osteogenesis Imperfecta," *Brit Journ Surg*, 1923-24 II, 737
- OSTEIN, J. M., "Traité d'anatomie pathologique," 1833, II, 204 Paris: Lavyrault
- ROUK, W., "Tabule ad Illustrandam Embryogenese Homini et Mammalium, tam Naturalem quam abnormem," 1849 Amsterdam

### Rickets

- ROMFR, R. S., "Rickets," *Amer Journ Rad*, 1933, XXX, 582
- AYBELL, E. J., and CHICK, H., "Hunger Osteomalacia in Vienna," *Lancet*, 1921, II, 842
- AXWELL, J. P., "Further Studies in Adult Rickets and Fatal Rickets," *Proc Roy Soc Med*, 1934, XXVIII, 265
- IMBERGER, H., "A Study of Developing, Florid and Healing Rickets as Demonstrated by X-ray Photography," "Studies of Rickets in Vienna, 1919-22," *Med Res Council*, 1923, No 77, 95

### Osteomalacia

- AYBELL, E. J., and CHICK, H., "Hunger Osteomalacia in Vienna," *Lancet*, 1921, II, 842
- EDENSKY, K., "Heilung von Rachitis durch Kunstliche Höhen-sonne," *Deutsche med Woch*, 1919, I, 712
- UNTER, D., "Calcium and Phosphorus Metabolism," *Lancet* 1930 I, 897, "Studies in Calcium and Phosphorus Metabolism in Generalized Diseases of Bones," *Proc Roy Soc Med*, 1935 XXVIII, 66
- OSER, E., "Über Spätrachitis und Osteomalacie Klinische, roentgenologische und pathologisch-anatomische Untersuchungen," *Deutsch J Chir*, 1920, CLII, 210
- AXWELL, J. P., "Further Studies in Osteomalacia," *Proc Roy Soc Med*, 1929-30, I, 639
- AXWELL, J. P., and MILLS, L. M., "Osteomalacia in China," *Journ Obst and Gynec Brit Empire*, 1925, XXXII, 433

- MELLANBY, E., "Accessory Factors in Experimental Rickets" *Journ Physiol* 1918-19  
 LII, *Proc* XI, "Part played by Accessory Food Factors," *Journ Physiol*,  
 1918 19, LII, *Proc* LIII
- MILKMAN, L. A., "Pseudofractures (Hunger Osteopathy, Late Rickets, Osteomalacia)  
*Amer Journ Roentgenol*, 1930, XXIV, 27
- POMMER, G. "Untersuchungen über Osteomalacie und Rachitis" 1885 Leipzig

### Idiopathic Steatorrhœa

- BENNETT, I., HUNTER, D., and VAUGHAN, J., 'Idiopathic Steatorrhœa (Gee's Disease)',  
*Quart Journ Med*, 1932, I, 603
- GEE, S., 'On the Caline Affection, *St Bartholomew's Hosp Rep* 1888 XXIV, 17
- HUNTER, D., 'Studies in Calcium and Phosphorus Metabolism in Generalized Diseases  
 of Bones, *Proc, Roy Soc Med*, 1935 XXVIII, 68
- SNFELL, A. M., and CAMP, J. D., 'Chronic Idiopathic Steatorrhea,' *Arch Int Med*,  
 1934, LIII, 615

### Scurvy

- BROMER, R. S., "A Roentgen ray Diagnosis of Infantile Scurvy" *Amer Journ  
 Roentgenol*, 1928, XIX 112
- FRAENKEL, E., 'Untersuchungen über die Moller-Barlowsche Krankheit' *Fort a d  
 Geb d R ntgen*, 1906 X, I
- KATO, K., 'A Critique of the Roentgen Signs of Infantile Scurvy, with Report of  
 Thirteen Cases' *Radiology*, 1932, XVIII, 1096
- PFLKAN, K. F., 'Roentgenogram in Early Scurvy,' *Amer Journ Dis Child*, 1925  
 XXX, 174
- WIMBERGER, H., 'Zur Diagnose die Säuglingskorkbuts' *Ztschr f Kind* 1923 XXXI  
 279 'Die Spätdiagnose des Säuglingskorkbuts,' *Fort a d Geb d R ntgen*, 1924  
 XXXVII 17

### Gout

- PHILIPS, H., POMTRANZ, M. M., and PINCUS, J., 'Gout,' *Journ of Rad*, 1925 VI 89

### The Lipoid Granulomatoses

#### Xanthomatosis

- CHRISTIAN, H. A., 'Defects in Membranous Bones Exophthalmos and Diabetes  
 Insipidus Contrib to Med and Biologic Research 1919 I 390
- HAND, A., 'Defects of Membranous Bones Exophthalmos and Polyuria in Childhood'  
*Amer Journ Med Sc* 1921, CLXII, 509
- JANSSON, G., 'Selektveränderungen bei der Schuller Christian'schen Krankheit'  
*Acta Radiol* 1935 XVI 59
- MOKEAU, J., 'La Dysostose Hypophysaire, Maladie de Schuller,' *Arch Frisco Belys  
 de Chir*, 1930 XXXII, 865 and 961
- ROWLAND, R. S., 'Schuller Christian's Disease' *Amer Journ Roentgenol* 1933 XX,  
 649
- SCHULLER, A., 'Über eigenartige Schädeldefekte in Jungendalter,' *Fort a d Geb d  
 R ntgen* 1915 XXIII, 12
- SNAPPER, I., 'Ziekten van het skelet' 1937 Haarlem
- SNAPPER, I. and PARISEL, CHR., 'Xanthomatosis Generalisata Ossium' *Quart Journ  
 Med* 1933 II 407
- SOSMAN, M. C., 'Xanthomatosis' *Amer Journ Roentgenol*, 1930 XXIII 581
- WORTIS, S. B., WOLF, A., and DYKE, C. G., 'Xanthomatosis and the Syndrome of  
 Diabetic Exophthalmic Dysostosis,' *Amer Journ Dis Child*, 1936 II 373



*Gaucher's Disease*

- BLOEM, TH F, GROEN, J, and POSTMA, C, "Gaucher's Disease," *Quart Journ Med*, 1936, V, 517
- FISCHER, A W, "Das Röntgenbild der Knochen, besonders des Femur in der Diagnose des Morbus Gaucher," *Fort a d Geb d Rönt*, 1928, XXVII, 158
- GAUCHER, E, "De l'Épithélioma Primitif de la Rate Hypertrophie Idiopathique de la Rate sans Leucémie," 1882 Paris Octave Doin
- KIRKLIN, B R, and HEFKE, H W, "Roentgenologically Demonstrable Changes in Bone in Gaucher's Disease. Reprint of a Case," *Amer Journ Roentgenol*, 1930, XXIV, 258
- PICK L, "Zur pathologischen Anatomie des Morbus Gaucher," *Med Klin*, 1922, XVIII, 1923, "Classification of Diseases of Lipoid Metabolism and Gaucher's Disease," *Amer Journ Med Sc*, 1933, CLXXV, 453
- REISS, O, and KATO, K, 'Gaucher's Disease,' *Amer Journ Dis Child*, 1932, XLIII, 365
- WORTH, H M, 'A Case of Gaucher's Disease,' *Brit Journ Radiol*, 1936, IX, 753

*Niemann Pick's Disease*

- PICK L, 'Niemann Pick's Disease and Other Forms of so-called Xanthomatosis,' *Amer Journ Med Sc*, 1933, CLXXV, 601

*Hyperparathyroidism*

- DRESSER, R 'Osteitis Fibrosa Cystica,' *Amer Journ Roentgenol*, 1933, XXX, 596.
- HUNTER, D, 'Calcium and Phosphorus Metabolism,' *Lancet*, 1930, I, 947, 'Calcium and Phosphorus Metabolism,' *Quart Journ Med*, 1931, XXIV, 393, 'Hyperparathyroidism,' *Proc Roy Soc Med*, 1931, XXIV, 40, 'Studies in Calcium and Phosphorus Metabolism in Generalized Diseases of Bones,' *Proc Roy Soc Med*, 1935 XXVIII, *Clin Sect*, 56
- HUNTER, D, and TURNBULL, H M, 'Hyperparathyroidism,' *Brit Journ Surg*, 1931, XIX, 203
- SNAPPEER, I 'Ziekten van het skelet' 1937 Haarlem

*Thyrotoxic Osteoporosis*

- AUB J C, BAUBER, W, HEATH, C, and ROPES, M, 'Studies of Calcium and Phosphorus Metabolism,' *Journ Clin Invest*, 1929, VII, 97
- HUNTER, D, 'Calcium and Phosphorus Metabolism,' *Lancet*, 1930, I, 947, 'Studies in Calcium and Phosphorus Metabolism in Generalized Diseases of Bones,' *Proc Roy Soc Med*, 1935, XXVIII, *Clin Sect* 60
- SNAPPEER, I, 'Ziekten van het skelet,' 1937 Haarlem

*Infantilism*

- ELLIS, A W, and EVANS, H, 'Renal Dwarfism,' *Quart Journ Med*, 1933, II, 231
- HASTINGS, G, 'Disorders of Post natal Growth and Development,' 1911 London Adlard & Son
- LOOSER, E, 'Die Kretinenhufte' *Schweiz med Woch*, 1929, X, 1258, 'Über die Ossifikationsstörungen bei Kretinismus,' *Verhandl Deutsch Path Ges*, 1929, XXIV, 352
- PARSONS, L, and BARLING, S, 'Diseases of Infancy,' 1933, I, 277 London Oxford University Press
- SCHINZ, H K, Personal Communication
- TFALL, C G 'Radiological Study of Bone Changes in Renal Infantilism' *Proc Roy Soc Med*, 1927, XXI, 717, 'Radiological Study of Bone Changes in Renal

Infantilism *Brit Journ Rad* 1928 XXI 25    The Radiological Appearance of Certain Bone Dyscrasias in Infancy and Childhood *Proc Roy Soc Med* 1934 XXVII 1385

VOGT F G    Renal Rickets *Amer Journ Roent* 1933 XXX 624

### Precocity and Giantism

BROOKE BLAND P and GOLDSTEIN L    Pituitary Basophilism *Surg Gynec and Obst* 1938 XXII 374

BROSTER L R and VINES H W C    The Adrenal Cortex 1933    London    Lewin

CAHILL G T    Air Injections to Demonstrate the Adrenals by X rays *Journ Urol* 1935 XXIV 238

CROOKE A C    Changes in Basophil Cells of the Pituitary Gland Common to Conditions which Exhibit the Syndrome Attributed to Basophil Adenoma *Journ Path and Bact* 1935 XLI 339

CUSHING H    The Basophil Adenomas of the Pituitary Body *Bull Johns Hopk's Hsp* 1932 L 137    The Pituitary Body Hypothalamus and Parasympathetic Nervous System 1932    Springfield Ill    C C Thomas

DAVIDOFF I M    Studies in Acromegaly *Endocrinol* 1926 X 461

JUPE M H    Discussion on Skeletal Changes in Metabolic and Endocrine Disorders *Proc Roy Soc Med* 1938 XXXI 198

LE MARQUAND H S and RUSSELL D S    Case of Pubertas Præcox (Macrogenitosomia Præcox) in Boy associated with Tumour in the Floor of the Third Ventricle *Roy Berks Hosp Rep* 1934 25 31

LEYTON O TURNBULL H M and BRATTON A B    Primary Cancer of the Thymus with Paraglandular Disturbance *Journ Path and Bact* 1931 XXXIV 63a

RUSSELL D S EVANS H and CROOKF A C    Two Cases of Basophil Adenoma of the Pituitary Body *Lancet* 1934 II 240

### Lead Poisoning

ROGERS T S PECK J R S and JUPE M H    Lead Poisoning in Children *Lancet* 1934 II 129

VOGT F G    A Roentgen Sign of Plumbism *Amer Journ Roentgenol* 1930 XXII 5a0

### Kashin-Bek's Disease

BEK I V    Arthritis Deformans in the Province beyond the Baikal *Russk Vrach* 190 V 74 (Quoted by PIRIE A H *Amer Journ Roentgenol* 1933 XXX 621)

### Hypertrophic Osteoarthropathy

KENNEDY R L    Hypertrophic Pulmonary Osteoarthropathy *Amer Journ Dis Child* 193 LIV 795

### Psoriasis Arthropathica

GARROD A and EVANS G    Arthropathia Psoriatica *Quart Journ Med* 1934 XXII 111

### Blood Diseases

BATY J M BLACKFAN K D and DIAMOND I K    Blood Studies in Infants and in Children *Amer Journ Dis Child* 1932 LVIII 667

BORZEL F F    Erythroblastic Anemia *Amer Journ Roent* 1933 XXX 6a

BUSH C B    A Case of Cooley's Anemia *Brit Journ Rad* 1937 X 491

COOLEY T B and LEF P    Erythroblastic Anemia *Amer Journ Dis Child* 1934 LVIII 70a

- GRINMAN, A. G., "Roentgenologic Bone Changes in Sickle Cell and Erythroblastic Anemia," *Amer Journ Roentgenol*, 1933, XXXIV, 297
- VOGT, E. C., and DIAMOND, L. K., "Congenital Anemias, Roentgenologically Considered," *Amer Journ Roentgenol*, 1930, XXIII, 625

### Osteosclerotic Anæmia

- CHAI MAN, E. M., "Osteosclerotic Anæmia," *Amer Journ Med Sc*, 1933, CLXXXV, 171
- HUNTER, D., "Studies in Calcium and Phosphorus Metabolism in Generalized Diseases of Bones," *Proc Roy Soc Med*, 1935, XXVIII, 64
- OVERGOOD, H., "Osteo-sclerotic Anæmia," *Acta Radiol*, 1936 XVII, 51
- VUIGHAN, J., "The Anæmias," 1936 London Oxford University Press
- VUIGHAN, J. M., "Leuco-erythroblastic Anæmia," *Journ Path and Bact*, 1936 XLII, 541

### The Leukæmias

- BATI, J. M., and VOGT, E. C., "Bone Changes of Leukemia in Children," *Amer Journ Roentgenol*, 1935, XXXIV, 310
- CARVER, L. F., and COPELAND, M. M., "Changes of the Bones in the Leukemias," *Arch Surg*, 1935 XXX, 630
- TAYLOR, H. K., "Periosteal Changes in a Case of Lymphatic Leukæmia," *Radiology*, 1926, VI, 523

### Hodgkin's Disease

- CARVER, L. F., and COPELAND, M. M., "Changes in the Bones in Hodgkin's Granuloma," *Arch Surg*, 1934, XXVIII, 1062
- DRESSER, R., and SPENCER, J., "Hodgkin's Disease and Allied Conditions of Bone," *Amer Journ Roentgenol*, 1936 XXXVI, 809

### Carcinomatosis

- HUNTER, D., "Studies in Calcium and Phosphorus Metabolism in Generalized Diseases of Bones," *Proc Roy Soc. Med*, 1935, XXVIII, 63
- PINEY, A., "Carcinoma of Bone Marrow," *Brit Journ Surg*, 1922 X, 233

### Leontiasis Ossea

- DIDE, M., and LECLERC, P., "Contribution à l'Étude Radiographique de la Leontiasis Ossea," *Bull et Mem Soc de Radiol m'd de France*, 1936, XXIV, 56
- FREUND, L., "Idiopathic Familial Generalized Osteophytosis," *Amer Journ Roentgenol*, 1938, XXXIX, 216
- SNAPPE, I., "Ziekten van het skelet," 1937 Haarlem

### Osteopetrosis

- ALBERS SCHONBERG, H. (*Aer II Verein*, Hamburg, Feb 9, 1904) *Munch med Wochenschr*, 1904 LI, 365, "Eine bisher nicht beschriebene Allgemeinerkrankung des Skelettes im Röntgenbild," *Fortschr a d Geb d Röntgen*, 1907, XI, 261
- KARNHNER, R. G., "Osteopetrosis," *Amer Journ Roentgenol*, 1926, XVI, 405
- MCPHEE, C. V., "Osteopetrosis," *Amer Journ Roentgenol*, 1936 XXXVI, 816
- PEHU, M., POLICARD, A., and DEFOUR, A., "L'Osteopetrose, Ou Maladie des os Marbreux," *Presse Med*, 1931, XXXIX, 993
- PIRIE, A. H., "The Development of Marble Bones," *Amer Journ Roentgenol*, 1930, XXIV, 147, "Marble Bones," *Amer Journ Roentgenol*, 1933, XXX, 618
- RENDICH, R. A., and SHAFER, A. V., "Osteitis Condensans Illi," *Journ Bone and Joint Surg*, 1936, XVIII, 899
- SHAFER, J., "Osteitis Condensans Illi," *Lancet*, 1938, II, 1229
- STENSTROM, B., "Über Marmer-skelette," *Acta Radiol*, 1936, XVII, 140

## Melorheostosis

- LEFRI, A, and JOANNY, J, "Hyperostosis," *Bull et mem Soc med d hôp de Par*, 1922, XLVI, 1141  
 MOORE, J J, and LORIMIER, A A DE, "Melorheostosis Leri," *Amer Journ Roentgenol*, 1933, XXIX, 161  
 NATVIG, P, "Case of Melorheostosis," *Acta Radiol*, XVII, 498.

## Osteitis Fibrosa (Focal)

- HUNTER, D, "Studies in Calcium and Phosphorus Metabolism in Generalized Diseases of Bones," *Proc Roy Soc Med*, 1935, XXVIII, 59

## Hæmophilia

- BAETJER, F H and WATER'S C A, "Injuries and Diseases of the Bones and Joints," 1921 London H K Lewis  
 BULLOCH, W, and FILDES, P, "Treasury of Human Inheritance," V and VI, *Eng Lab Mem* 1911, XII  
 KLASON, T E, "Hæmophilia and Hemophilic Arthropathy," *Acta Radiol*, 1921, I, 26  
 PRIPS BUTTS C E, "Articular Changes in Hæmophilia," *Acta Radiol*, 1935 XVI, 502

## Osteitis Deformans

- BRAILS福德, J F, "Paget's Disease of Bone," *Brit Journ Radiol*, 1938, XI, 507  
 HUNTER, D, "Studies in Calcium and Phosphorus Metabolism in Generalized Diseases of Bones," *Proc Roy Soc Med*, 1935, 61  
 KAY, H D, SIMPSON, L S, and RIDDOCH, G, "Osteitis Deformans," *Arch Int Med*, 1934, LIII, 208  
 KNAGGS L, "On Osteitis Deformans (Paget's Disease) and its Relation to Osteitis Fibrosa and Osteomalacia," *Brit Journ Surg*, 1925-26, XIII, 206  
 SNAPPER I, "Ziekten van het skelet," 1937 Haarlem

## Atrophic Conditions of the Digits

- BARSONY T, and FRISCH, E, "Beitrage zur Rontgenologie der 'Akrosklerose,'" *Fortschr a d Geb d R ntgenstrahl*, 1933, XLVII, 287  
 BROWN G E, "Thrombo angustis obliterans," *Surg Gynec and Obst*, 1934 LVIII, 29  
 BUERGER, L, "Circulatory Disturbances of the Extremities," 1924, 214 Philadelphia W B Saunders Co  
 COLLINS, J N, "Idiopathic Hemorrhagic Sarcoma," *Amer Journ Roentgenol*, 1931, XXVI, 260  
 DE TAKATS, G, and MACKENZIE, W D, "Diagnosis and Treatment of Circulatory Disturbances of the Extremities," *Surg Gynec and Obst*, 1934, LVIII, 655  
 MAYO, W J, and ADSON, A W, "Raynaud's Disease, Thrombo Angustis Obliterans and Scleroderma," *Ann Surg*, 1932, XCVI, 771  
 PODKAMINSKY, N A, "Acrosclerosis Hyperplastica Intraossea," *Amer Journ Roentgenol*, 1937, XXXVIII, 889

## PART THREE

### SECTION IX

## TUMOURS AND CYSTS OF BONE

BY

R. E. ROBERTS, B.Sc., M.D., F.R.C., D.P.H., D.M.R.C.

### CHAPTER XLV

#### SIMPLE TUMOURS OF BONE

##### GENERAL CONSIDERATION

WHAT IS meant by a bone tumour? Broadly speaking, one implies any tumour which is derived from, or which produces, or is produced by bony tissue, or more correctly any tumour affecting bone, which originates from the primitive mesenchyme which is the common predecessor of bone, cartilage or fibrous tissue.

Our list comprises osteomata, osteochondromata, chondromata, giant cell tumours, metastatic tumours in bone, osteogenic sarcomata, Ewing's tumour, endotheliomata, myelomata, angiomas, chloromata, lymphomata, mesosarcomata, lipomata and neurogenic sarcomata.

##### OSTEOMATA AND OSTEOCHONDROMATA

The indiscriminate use of the term "exostosis" for bony outgrowths has caused considerable confusion for this term has been employed for such tumours regardless of whether they are pure bony outgrowths or whether cartilage plays a part in their constitution or growth. It therefore seems desirable to abandon the term "exostosis" and to speak of such tumours as either osteomata or osteochondromata, according to their composition.

##### OSTEOMATA

Osteomata may be found at any age. They may develop from any bone but their commonest sites are in connection with the bones of the skull, sinuses, orbit, jaws and the terminal phalanx of the great toe.

The associated symptoms are pain from pressure on soft tissues, and sensory or even motor disturbances from pressure on nerves. In the sinuses they are frequently of the nature of accidental discoveries, and symptomless.

Pathology.—They are bony tumours derived from subperiosteal osteo-

blasts As such they may be (i) simple or idiopathic or (ii) traumatic (the erroneously called myositis ossificans traumatica )



FIG 486—Intracranial osteoma Lateral ventricle filled with air

### SIMPLE OSTEOMA

**Radiological Appearances**—These tumours appear as projections from the surface of the bone Often of a dense homogeneous consistency they usually contain little or no cancellous internal structure In connection with the frontal and less frequently with the other accessory sinuses or the orbit they are usually rounded in contour In relation with the inner table of the calvarium they may show either a smooth flat or rounded contour (Fig 486) or else a wavy outline Their occasional association with meningiomata must be borne in mind In such cases the neurological clinical picture is of course of paramount importance



FIG 487—Simple frontal osteoma (exostosis) Note cancellous bone in interior

They are occasionally seen in relation to the outer table of the calvarium when they present a flat or rounded contour If growing from the mandible they are smooth and rounded in outline and of dense opacity In the terminal phalanges of the great toe they are often of a square contour less dense and usually contain cancellous bone in their interior (Fig 487)

In the rare variety known as 'endosteoma' growth of the osteoma is entirely *within* the marrow cavity

## TRAUMATIC OSTEOMA

This term is coined to include many of those bony outgrowths which for many years have appeared under the erroneous title of 'traumatic myositis ossificans'

Pathology — *Fay* designates the condition as a 'traumatic periosteal bone formation' and discusses its pathology very fully. *Lewis* and *Macewen* were both fully alive as to its true nature



FIG. 488.—Ossifying hematoma (so called myositis ossificans traumatica)



FIG. 489.—Traumatic osteoma (osteoma on an subperiosteal hematoma) resulting from a severe kick

The nature of this type of pathological ossification is as follows

Due either to a subperiosteal hæmorrhage resulting from a severe blow or to avulsion of the periosteum from the sudden pull of a strong tendon or to the gradual but progressive pull of an oft repeated occupational strain the periosteum becomes elevated from the bone. Into the subperiosteal hæmatoma so formed there is an ingrowth of fibroblasts and vessels, the former being

derived from perosteum tend to form bony matrix as well as ordinary connective tissue with the result that calcification readily occurs

**Radiological Features**—At first the radiographic appearances consist of faint calcified shadows possibly at some distance from the bone which gradually becomes more pronounced and spread in a veil like manner towards the bone (Fig 488) The calcification may extend right down to the bone or a gap may be present Ossification rapidly takes place in the calcified area and a bony mass results

Instances of this type of pathological ossification may be found in the region of the elbow after dislocation in the footballer's thigh after a severe kick (Fig 489) in the rider's bone of cavalry men above the patella after forcible avulsion of the quadriceps extensor and in other sites where the bone is subjected either to direct trauma or to forcible muscular avulsion The condition is commoner in the region of the elbow because dislocations are more frequent in this joint than in others and because dislocations in this region are invariably associated with avulsion of muscles

At the knee joint the quadriceps may be avulsed from the patella a subperiosteal hæmatoma may develop and a traumatic osteoma may be found above the patella through ossification in this hæmatoma If the joint is fixed in extension this new bone fuses with the patella if movement is allowed or practised a gap may occur between the ossified hæmatoma and the upper border of the patella

It should be noted that these traumatic osteomata attain their maximum size early and remain stationary for some time As a rule they diminish in size after a period and may eventually disappear

Allied to the traumatic osteomata or ossified subperiosteal hæmatomata are the bony spurs seen in the tendinous attachments of the triceps to the olecranon in the attachment of the tendo Achillis or of the short muscles of the foot to the os calcis and in many other similar sites Though there may be no history of forcible muscular strain in such cases there may be as *Leavis* suggests inco-ordinated muscular contractions which result in periosteal stripping Such bony spurs are often developed as a result of occupational strain due to repeated mild traumata

### OSTEOCHONDROMATA AND CHONDROMATA

In spite of the fact that both single and multiple exostoses or osteochondromata and chondromata are to be regarded as aberrations of development in the cartilaginous growth disc it seems reasonable to include them in the group of bony tumours Their structure and their mode of growth appear clearly to give them a right of place in such a category For the sake of completeness they are therefore included in this group even at the risk of duplication The frequent co-existence of multiple exostoses and of multiple chondromata in the same patient might indicate that they should be considered concurrently



but with the knowledge of such a co-existence in mind, and with the admission that they may be regarded in some cases as variants of the same pathological or aberrant developmental process, it seems expedient to regard them individually in this account.

### OSTEOCHONDROMATA

Owing to the close embryological connection between fibrous tissue, cartilage and bone, and their interchangeability under pathological conditions, an osteochondroma may arise in connection with any portion of bone or cartilage, or occasionally of fibrous tissue. It is a tumour whose radiological appearance depends largely on the relative proportions in it of opaque bone and translucent cartilage (with fibrous or myxomatous tissue) and of the distribution in it of these elements (Fig 490)

Apart from the osteochondromata developing from the bony parts, it should be noted that these may develop from the inner surface of the synovial membrane of joints, some of the ends may become detached and form loose bodies in the joints ("osteochondromatosis" of the knee and elbow joints)

Attention will be concentrated here on types of osteochondroma which are

associated with the diaphyseal cartilage of bones, namely the so-called "single exostosis" and the condition which for many years has been known as "multiple exostoses" but which will probably in future appear under the more correct title of "diaphyseal aclasis" or of "hereditary deforming chondrodysplasia"

#### Single Exostosis

Sites —Near the ends of a long bone, more particularly the femur, tibia, and humerus. Cases have been recorded where a familial history has been traced.



FIG 490.—Osteochondroma of iliac bone. About equal proportions of bone and cartilage

**Clinical Features** —These tumours may give rise to pain but unless a fracture of the exostosis has occurred (Fig 491) this is usually due to pressure on overlying soft tissues or to inflammation in the overlying bursa which is

frequently present or to superimposed malignant changes in the tumour. Trauma rather than being a cause of such tumours is frequently the factor which brings about their discovery the patient being examined because of pain or dysfunction.

**Pathology** —Unfortunately owing to the paucity of pathological data views expressed as to the origin of these tumours are based on theory rather than on fact.

(1) Some authors are of opinion that these exostoses being frequently found at the site of the attachment to bone of powerful muscles or tendons such as the adductor magnus are related to an abnormal development of the pre-cartilaginous tissues which such tendons during the course of their development normally contain at their osseous attachment. In other words they would be regarded as exaggerated tubercles for muscular attachments. If this were the case one would expect them to develop

solely along the lines of muscular tension occasionally however they not only erode neighbouring bones but have been known to turn in and invade even the parent bone.

(2) Another theory is that they are relics of the embryonic connective tissue the blastema from which joints are developed in embryo. If this view were correct the cartilaginous cap of the exostosis would be of the nature of articular cartilage rather than of metaphyseal cartilage. In their mode of growth however these exostoses bear a much closer resemblance to that of metaphyseal cartilage than to that of articular cartilage.

(3) A more reasonable explanation is that during the process of growth a portion from the edge of the metaphyseal cartilage becomes detached from the main cartilage and displaced on to the surface of the shaft and that this separate fragment continues to grow independently at right angles to the main line



FIG 491 Single exostosis (osteochondroma) of femur with fractured pedicle

of growth of the bone. The position of the exostosis in relation to the centre of the shaft will therefore depend on the time during the growth of the bone when the diaphyseal fragment became left behind. The cartilaginous cap, behaving as an independent metaphysis, causes growth of the exostosis, whilst its inclination to the parent bone may be attributed partly to muscular or aponeurotic tension or pressure, and partly to continued growth of the main bone on the epiphyseal side of the exostosis.

Unless malignancy supervenes, growth of the exostosis normally ceases at the same time as metaphyseal growth in the parent bone ceases; the cartilaginous cap then becomes ossified.

It should be noted that in cases of "multiple exostoses" or diaphyseal aclasis, an individual exostosis may present all the appearances of a "single exostosis" as described above. There appears to be no reason to infer that the formation of a single exostosis is in any way different from that of the more widespread lesion, except that in the former, one bone only is affected. In other words, the theory put forward by *Keith* to account for the formation of "multiple exostoses" is equally applicable, only in a more localised way, to the "single exostosis." *Hume* supports this view.

**Radiological Appearances.**—In the radiogram these tumours are shown as either sessile or pedunculated outgrowths from the bones on the diaphyseal side of the epiphyseal line. The ends of the tumours are almost invariably bigger and broader than their shafts. The free end of the exostosis is capped by cartilage, which, unless calcified or ossified, is not seen in the radiogram. The appearances of the free end vary according to the relative proportions of cartilage and bone which are present in it; in some cases it is small and smooth, in others large, cauliflower-shaped, and of irregular density. Like many other pathological collections of cartilage cells, this cartilaginous cap is liable to undergo malignant changes.

**Multiple Exostoses (Diaphyseal Aclasis, or Hereditary Deforming Chondrodysplasia).**—The varying titles given to this condition are a tribute to the views held as to its pathology.

**Age.**—This condition, which is hereditary and which may affect several individuals of the same family, may be discovered at any age period, but usually makes itself manifest during the period of active growth.

**Sites.**—It affects only those bones where bone laid down in cartilage comes to be covered by periosteal bone, such as the long bones or the scapula. The parts of the bones most affected are those parts where growth is normally greatest. It does not affect bones formed entirely within cartilage, and is therefore not found in the tarsal or carpal bones, nor in the sternum, nor in the epiphysis of long bones. Nor does it affect bones developed in membrane, such as those forming the cranial vault.

**Clinical Features.**—The more common clinical features of this condition are a shortening of stature, with bowing of the forearms and legs. Though the

disease is usually roughly symmetrical the limbs may be of different length. The phalanges may be deformed.

Palpable tumours of varying sizes are usually found in the affected parts and pain and dysfunction may result from pressure of the growth. This pressure may be so great as to cause paraplegia, local paralysis or even aneurysm through pressure of the tumours on the spinal cords or on nerves or on vessels. Local exacerbation of symptoms in patients over 30 years of age may be the first indication of malignant developments.

**Pathology**—In attempting to explain the pathological processes involved in this condition the theories propounded by *Keith* hold pride of place. These are a natural corollary to the discoveries of *John Hunter* concerning the growth of bones. The latter envisaged this growth as involving a double process.

(a) The deposition of new bone in the cartilaginous growth disc (the metaphysis) at the ends of the shafts. (b) A modelling process by which the new bone thus laid down is pruned, reformed and incorporated as an integral architectural part of the cylindrical shaft.

From these premises *Keith* deduced that in the condition which he named diaphyseal achasia the essential factor is a retardation or even arrest of the modelling process, deposition of new bone occurring at the same time. This would satisfactorily account for the known distribution of the disease in the skeleton.

Normally the periosteum is attached to the epiphyseal edge of the cartilaginous growth disc. As the bone grows the periosteal cuff of the growth disc extends with the lengthening bone so that the disc is always normally covered with periosteum or perichondrium. One of the main functions of this periosteal cuff and of the osteoclasts and osteoblasts derived from it is to model the growing cylinder of bone in such a manner that it maintains its normal shape. If for some reason the periosteal extension is arrested, areas of cartilage formed bone are exposed on the surface of the shaft and are free to expand in abnormal directions with the consequent formation of irregular outgrowths or exostoses.

In addition to the arrest of the periosteal sheath *Keith* thinks it likely that there is an irregular grouping and division of the cartilage cells. *Jansen* amplifies this suggestion. The aberrant growth of these cells would account for the usual shortening, epiphyseal tilting and occasional lengthening of the bone which is found in this disease.

According to *Wolff's* law the development of a bone depends on the stresses and strains to which it is subjected. This may account for the fact that in this disease the radius and tibia are as a rule better developed than the ulna and fibula, any stunting of growth which occurs being usually found in the latter bones.

The site of the exostosis on the shaft of the bone will presumably depend

on the degree of impairment of the circumferential modelling cuff and the age at which this, and the disorderly behaviour of the cartilage cells, occur. The earlier this onset occurs, the nearer to the centre of the shaft of the bone will be the exostosis. The impairment of bone modelling, according to *Keith*, may only be temporary.

If an exostosis occurs at or near the site of strong muscular attachment, it is only reasonable to expect that muscular strains will have some effect on the future shape of that exostosis.

*Keith* suggests that the process described may be due to an endocrine disturbance, probably a dysfunction of the thyroid gland. *Hume* suggests that it may be due to a defect in the secretions of the pituitary or genital glands because of occasional association of the disease with gigantism. *Jansen* suggests that it is due to a disturbance of the sympathetic nervous system.

**Radiological Appearances.**—Radiologically, the affected bones show, either individually or collectively, appearances which are characteristic. The metaphyseal ends of the bones are broadened and their internal structure grossly changed. Multiple osteochondromata, or so called "exostoses," usually with broad bases and pointed tips, are seen. (These are, as a rule, covered from their tips down the slope towards the epiphyseal line by cartilage, of this cartilage we have, of course, no X ray evidence unless it becomes calcified.) The cortex is thin and the medullary spaces wide, irregular, and translucent. The bones of the forearm and lower leg are bent, the ulna and fibula are usually shortened, and subluxations at the wrist, elbow, or ankle are frequently present. The epiphyseal axis is often distorted.

Where the osteochondromata or exostoses protrude between adjacent bones, such as the tibia and fibula, one or other of these bones may show absorption from erosion, or local fusion may occur.

## CHONDROMATA

These cartilaginous growths, occurring usually between the ages of 20 and 30 years, may be either single or multiple.

### Single Chondroma

**Sites.**—The single chondroma occurs most frequently in a small long bone of the hand or foot, in a rib near the costal cartilage, in the sternum, pelvic bones, scapula, occasionally in the spine, and rarely in the skull. There is often a history of preceding trauma.

**Radiological Appearances.**—Radiologically, the appearance of a chondroma varies according to whether it has arisen in the central or the peripheral portion of the affected bone. If centrally situated it is shown as a well defined translucent area surrounded by a bony capsule (Fig. 492), if the chondroma is large, the cortex may be thinned and expanded, trabeculation may be present

in the translucent area. *If arising in the cortical bone* the latter is destroyed so as to present a depression in its surface whilst the more superficial aspect of the tumour is seen as a round faint shadow protruding into the soft tissues and deforming the outline of the affected part. Whenever the growth has perforated the surface of bone or penetrated into the soft tissues relics of the original cortical covering are seen as bony flakes of varying sizes on the peri-

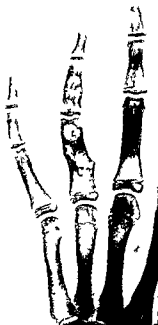


FIG 492—Central chondromata of phalanges with a pathological fracture. Note encapsulating sclerotic area in the bone



FIG 493—Eccentric chondroma showing flakes of cortex on surface of cartilaginous tumour

phery of the growth (Fig 493). Sometimes chondromata contain areas of calcification and ossification in their interior.

Pathological fractures, though reputed to be less frequent than in simple bone cysts, are frequently seen; they are more likely to occur if myxomatous degeneration has taken place in the chondroma.

In the hand and foot these tumours are more common in the phalanges than in the metacarpals or metatarsals—the reverse of the distribution in these parts of bone cysts and benign giant celled tumours.

In the long bones single chondromata are comparatively rare.

Malignant changes may supervene in this, as in all other growths where cartilage predominates.

### Multiple Chondromata

**Sites.**—These have the same anatomical distribution as single chondromata ; they may, however, also be found in the long bones, such as the humerus, radius, femur, or tibia.

Because of their occurrence near the epiphyseal lines of the long bones

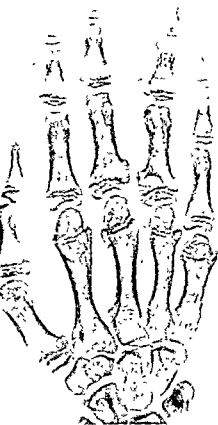


FIG. 494.—Multiple exostoses confined to metaphyseal regions of metacarpals and phalanges.



FIG. 495.—Multiple chondromata in a case of diaphyseal aclasis.

(Fig. 494), considerable confusion has arisen regarding the mutual relationship of multiple exostoses and multiple chondromata.

**Pathology.**—*Hume* regards the chondroma as being due to an abnormal stimulus affecting the centre of the growth-disc alone, and interfering with the process of ossification ; a single exostosis is regarded as being due to a stimulus affecting a localised area of the periphery ; while multiple exostoses are regarded as being due to a similar but more general stimulus, affecting the growth-disc of all the long bones. *Keith* also regards the two diseases (multiple



FIG 496 —Multiple ossifying echondromata of the hand

exostoses and multiple chondromata) as being of a similar nature *Jansen* groups the two conditions under a common heading and emphasises their relationship

Cases are not infrequently seen in which both multiple exostoses and multiple chondromata co exist (Fig 495)

**Radiological Appearances**—The X ray appearances of the individual tumours resemble these already described under single chondromata (central or eccentric)



### Ollier's Disease

Ollier has described a unilateral variety of chondro-dysplasia occurring in children (subsequently the changes in this disease have often been found to be bilateral). The distribution of the lesions in this disease follows closely that encountered in multiple exostoses; this has led Keith and others to conclude that Ollier's disease is merely a variety of diaphyseal aclasis.

**Radiological Appearances**—In this disease there may be present the lesions either of multiple exostoses or of multiple chondromata or of both. In addition some of the bones show characteristic appearances in the nature of longitudinal striae of translucency surrounded by lines of increased density. The nutrient foramina may be enlarged; the translucent lines running from the enlarged foramina towards or even up to the epiphyseal line. There may also be stippling in the carpal and tarsal bones and occasionally in the metaphyses and epiphyses of the long bones and in the pelvis, scapula and clavicle (osteopoikilic). In any individual case one or other of these appearances may be the outstanding feature. As the patient reaches adult life some of the phenomena become less obvious and may even disappear.

**Pathology**—Jansen lays stress on the fact that in this disease there are marked evidences of disturbances of the blood vessels or of their innervation. Bent on in experiments on rabbits has been able to reproduce some of the characteristic appearances of Ollier's disease such as the unossified cartilage and the striation by injuring the nutrient artery to the tibia by injection of 96 per cent alcohol. He also points out that the dense metaphyseal stria and the radiating striae in the iliac bone follow the lines of the blood vessels. He concludes that paresis of the blood vessels is the cause of the disease. From this Jansen whilst adhering to the theory that the appearances are due to a dissociation of bone growth regards the latter as being brought about either by an insufficient formation of blood vessels in the affected parts or by a vasoconstriction due to faulty action of the sympathetic nervous system.

There is obviously a close relationship between diaphyseal aclasis multiple



FIG. 437.—Humerus of same case as Fig. 436. Chondromata of the humeral shaft not ossified.

enchondromata and Ollier's disease our knowledge of the cause of the processes involved is however still vague

### GIANT CELL TUMOUR (OSTEOCLASTOMA)

Though termed benign it should be noted that these tumours tend to recur locally after inadequate surgical treatment and cases have been recorded where lung metastases have occurred possibly due to the release into the blood stream of viable cells which have been dislodged during curettage

**Age Incidence**—This tumour is usually discovered between the ages of 20 and 40 years the epiphyseal line thus being as a rule closed



FIG. 498—Giant cell tumour of femur showing trabeculation. Note metaphyseal rather than epiphyseal site in this case

**Sites**—Usually in the epiphyseal or juxta-epiphyseal region of the lower end of the femur the upper end of the tibia or the lower end of the radius or tibia it may less frequently be found in the upper end of the fibula or radius or more rarely still in the mandible vertebrae or other bones

**Clinical Features**—Many cases give a definite history of local trauma followed by pain and later by tumour formation Trauma is a more specific antecedent or possible cause of these tumours than is the case in simple bone cysts In the latter the trauma is usually the factor which draws attention to the pre-existing cyst in the giant cell tumour the trauma is more likely to be the cause of the subsequent tumour

The pain in the giant cell tumour is more severe and more constant than in

**Simple cysts**—The rate of growth of the tumour, though slow, is more rapid in the giant-cell tumour but pathological fractures are less frequent than in bone cysts, though dysfunction from local deformity may be present.

Rarely the degree of vascularity of these tumours is so great that on palpation expansile pulsation is felt—one of the varieties of "bone aneurysm" being produced.

**Pathology**—The characteristic histological finding in these tumours is that of a large number of multinucleated giant cells many of which are uniform in size and in the size of their nuclei (in contradistinction to those found in osteogenic sarcoma in which the size of the nuclei varies). There is a connective tissue matrix and many thin walled capillaries which give rise to the areas of hemorrhage which are usually seen in the tumour. When the vascular tissue is very abundant, the so called "bone aneurysm" may be formed. The periosteum overlying the tumour shows a tendency to form new bone which serves to limit or encapsulate the tumour. The line of demarcation between the tumour and healthy bone is as a rule, sharply defined.

**Radiological Appearances** The affected portion of bone, either in whole or more commonly on one side only, shows an expansion surrounding an area of translucency, in which coarse trabeculae may or may not be seen (Fig 498).

The cortex is thin and may be broken, either by a spontaneous fracture, or, more rarely, by destruction with extension of the tumour into soft tissues (Fig 499). Where two bones are adjacent, the tumour may spread from one to the other along fascial or tendinous planes.

The tumour is not encapsulated by sclerosed bone as in the chondroma. It may extend up to, but not as a rule through, the neigh-



FIG 499—Giant-cell tumour of humerus. An appearance suggestive of palisade spicules is seen, and as it subsequently expanded and destroyed the cortex and extensively invaded the soft tissues a radiological diagnosis of osteogenic sarcoma was made. On microscope examination however it was reported to be a pure myeloma—the old name for giant-cell tumour.

bouring articular surface, the subarticular bone shows a slight reactive thickening and a sharp line of demarcation

The tumour taking the line of least resistance grows up the medullary canal but shows a sharp line of demarcation (unlike the sarcoma). In view of its dome like diaphyseal limit this may show in the radiogram a graded density at this periphery

Frequently the tumour is subcortical and confined to one side of the bone. In such cases a 'soap bubble' appearance on the surface of the bone may occasionally be presented

Where bony projections are present in the vicinity of the tumour (e.g. the femoral condyle or the adductor tubercle or the transverse process of a vertebra) the tumour tends to grow into such projections

### VARIANTS OF THE GIANT-CELL TUMOUR

Several pathologically atypical or modified varieties of giant cell tumour may occur. As these possess some points of radiological and prognostic importance the more important are described here

#### (1) Xanthosarcoma

Pathologically this is a solid type of giant cell tumour which is yellow in colour because of many large polyhedral cells containing yellow lipid granules (so called xanthomatous cells)

Radiologically it is often big and may break down its bony periphery. It grows into but does not infiltrate the soft tissues

It may destroy the epiphysis and bring about collapse of the joint surface thus differing from the typical giant cell tumour

It tends to recur after curettage and on becoming infected it may show the superimposed X ray appearances of an osteomyelitis

#### (2) Fibrosing Type of Giant-cell Tumour

In this part or whole of the tumour becomes converted into a spindle cell collagenous connective tissue. This appears to be a healing process, such a tumour may become a fibrous mass easily shelled out

Radiologically, there is no characteristic feature to distinguish this tumour from a giant cell tumour

#### (3) Giant cell Tumour of Cartilaginous Origin

This is merely a giant cell tumour appearing at the ends of long bones in which streaks or masses of cartilage near the epiphyseal line have been absorbed. It shows no characteristic X ray appearances

#### (4) Telangiectatic Giant cell Tumour

This is a non malignant tumour often extending a considerable distance along the length of the shaft

Expansion of the bone, with trabeculae or septa separating the individual translucent spaces, is seen.

On palpation expansile pulsation is felt, and both radiologically and even microscopically it may be difficult to differentiate it from the malignant bone aneurysm (telangiectatic osteogenic sarcoma). The latter is, however, more rapid in its growth, tends to absorb its peripheral walls more readily, produces lung metastases, and rapidly comes to a fatal termination.

#### (5) Border-line Cases of Giant-cell Tumour

These are tumours of doubtful benignity, which, in addition to giant cells, contain spindle cells, though the latter are not markedly deeply staining.

Central in location, they cause much bony destruction, perforate the cortex, and extend into the soft tissue. Radiologically, the chief differential point between them and osteogenic sarcoma is that they do not involve the periosteum, and in no view is a reactive triangle demonstrable.

They recur locally and should inspire a guarded prognosis.

#### (6) Apparently Secondary Giant-cell Structure in Malignant Osteogenic Sarcoma

Wherever bone is in process of absorption, giant cells of the foreign-body type are found. It is only to be expected that in osteogenic tumours where there is blood-clot or infection, or where there has been trauma (surgical or otherwise), such giant cells should be present. It is the finding of such cells, in the absence of the typical cells of osteogenic sarcoma, which may lead the pathologist to the erroneous diagnosis of giant-cell tumour in a case of osteogenic sarcoma.

This fact has led *Ewing* to insist that such histological examinations *per se* may be fatally misleading unless a typical portion of tumour tissue is removed and submitted for examination.

## HÆMANGIOMA OF BONE

In 1930 *Bucy* and *Capp* analysed and summarised the hitherto published cases of this condition, which, though a clinical rarity, is not infrequently found at post-mortem examination. They describe three types of case, each showing characteristic X-ray appearances.

#### (1) Hæmangioma of the Vertebrae

Occurring at any age, symptoms, when present, are those of compression of the spinal cord, and are indistinguishable from those of tumour of the spinal cord. The radiographic appearances are characteristic. Owing to an irregular absorption of some of the trabeculae, and thickening of others, the vertebral body shows parallel coarse vertical striations, with loss of normal architecture and an increased translucency of the bone (Fig. 500). The abnormal trabecular formation may involve the pedicles, transverse and spinous processes, the direction of the striæ varying accordingly. More than one vertebra may be affected. Collapse of the vertebral body is rare. The discs are not affected.

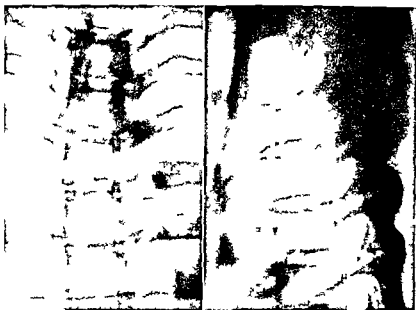


FIG. 500 Hæmangioma of a dorsal vertebra

## (2) Hæmangioma of the Skull Flat Bones and Clavicle

Though commonest in the skull this tumour is also found in the scapula humerus clavicle and occasionally in the long bones.

Radiographically there is evidence of expansion of the bone with loss of translucency. The tumour shows a honeycombed appearance with fine bony trabeculae radiating from its centre. The cortex may be penetrated or destroyed but the periosteum remains intact though expanded and forms a thin bony shell (cf. osteogenic sarcoma).

## (3) Hæmangioma of the Long Bones

The characteristic radiographic feature is an almost complete absence of erosion with the presentation of an appearance resembling soap bubbles in the subcortical or subperiosteal regions at the ends of the diaphysis. The X-ray appearances may resemble those of a giant cell tumour except that in a hæmangioma the loculae are smaller and are traversed by a fine fibrillary framework. The cortex is partially eroded, the periosteum is intact but expanded with the resultant formation of a thin bony capsule or shell. The tumour may affect one side of the bone or it may completely surround it.

Pathologically the tumour is usually of the nature of a cavernous hæmangioma with large blood-filled cavernous spaces and a variable amount of connective tissue stroma.

These tumours are benign and are responsive to X-ray treatment.

## CHAPTER XLVI

### MALIGNANT TUMOURS OF BONE

#### CLASSIFICATION OF MALIGNANT BONE TUMOURS

MANY CLASSIFICATIONS of these tumours have been evolved, some depending on their life-history, others on their pathological anatomy and their histological structure. The best known of such classifications is that adopted by the American Bone Registry. This concerns itself chiefly with the life-history of the tumours, relegating to a secondary position their histological structure.

Their classification is as follows :

- 1 Metastatic tumours, primary in tissues other than bone.
2. Periosteal fibrosarcoma.
- 3 Osteogenic tumours, (a) benign, (b) malignant.
- 4 Inflammatory conditions.
- 5 Benign giant-cell tumours.
- 6 Angiomata, (a) benign, (b) malignant.
7. Ewing's tumour.
8. Myeloma.

This classification, from the radiologist's point of view, holds pride of place, for it not only includes all the malignant tumours which he may meet, but also takes into consideration the inflammatory lesions and the benign tumours which may cause confusion in the differential diagnosis.

#### METASTATIC BONE TUMOURS

**Age Incidence.**—Metastatic carcinoma in bone is usually found after the age of 35 years, most commonly between the ages of 40 and 60 years.

✓ **Sites.**—These tumours are usually multiple, but may on occasion be solitary. *Any bone of the body may be involved, but they are not common below the knee and the elbow.* The commonest sites are the upper end of the femur and of the humerus, the vertebral bodies, the pelvic girdle, the ribs, and the cranial vault.

Metastases from a breast carcinoma (more frequently of the scirrhus type) are commonest in the spine, pelvis, femur, skull, ribs, and humerus. From a prostatic carcinoma the metastases are commonest in the pelvis and lumbosacral spine. From a hypernephroma common metastatic sites are the spine, pelvis, femur, humerus, skull, sternum, and ribs.

**Clinical Features.**—Though pain is a common symptom of these tumours it does not necessarily arise because of the bony involvement alone, but because

of involvement of nerves. The pain associated with spinal metastases arises as a rule only when nerve roots are involved. One has seen cases in which pain has been a prominent symptom months before the vertebral metastases have become demonstrable radiographically.

Not infrequently attention is drawn to the lesion in a long bone only by the occurrence of a spontaneous fracture without any preceding pain. A palpable



FIG. 501. Metastases from breast carcinoma in humerus, scapula and ribs. In spite of marked destruction, collapse has not yet occurred in the humerus.

tumour in a long bone is usually found only after a spontaneous fracture has taken place.

It should be noted that bony metastases may occur many years after the primary lesion has been discovered and dealt with.

**Pathology**—The pathology of the secondary tumour obviously depends on the nature of the primary growth.

The commonest primary sites are the breast (Fig. 501), the prostate, the adrenal glands (hypernephroma) (Fig. 502) and the thyroid. Less common



primary sites are the alimentary tract (including the tongue) (Fig 503) and the lung (Fig 504) and less common still the testicle (Fig 505) bladder, ovary, or skin. Histologically the metastasis shows the same structure as the primary growth.

Metastasis takes place more commonly via the blood stream (hence the frequent origin of metastases in the bone near the nutrient vessels) or occasionally via the lymphatics (such as those connecting the femur with the pelvis via the ligamentum teres).

Radiographically the metastasis may be "osteolytic," or it may be 'osteoblastic' or 'sclerosing' but frequently a combination of these is seen.



FIG 503—Metastasis in vertebral body with as yet only slight collapse (primary in tongue)



FIG 504—Solitary metastasis in humerus from hypernephroma. Collapse has not yet occurred.

Bence Jones proteoses may be discovered in the urine in cases of metastatic carcinoma.

**Radiological Appearances**—The X-ray appearances are dependent on the invasion of the bone by malignant cells deposited in it either by the blood stream or lymphatic channels. At first these deposits are too small to show any evidence in the radiogram, which may in such cases have been taken because of local pain in the bone. In cases of persistent pain in the bone without obvious radiographic cause it is therefore essential to repeat the examination at frequent intervals (Fig 506) because of the possibility of a developing metastatic or sarcomatous disease which is not demonstrable in the earlier radiograms.

Where metastases have occurred they will become radiographically evident within a few weeks. As the bone change is one of *replacement* of the invaded bone by neoplastic tissue and as there is as a rule little or no reactive subperiosteal proliferation no evidence of tumour formation in the long bone nor of collapse in the vertebral bodies is found in the early stages.

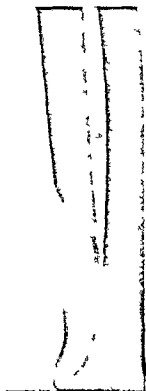


FIG 504—Metastases in ulna from primary bronchogenic carcinoma. Note punched out appearance of bone which has been observed in many metastases from lungs.



FIG 505—Metastases in tibia and astragalus from malignant teratoma of testicle.

**OSTEOLYTIC METASTASES**—The commonest metastases—the so-called osteolytic type—are not only composed of tissue which is more translucent than bone but they evoke no reaction in the nature of increased ossification. They are therefore shown in the radiograms as translucent areas in one or more bones irregular in shape and of varying sizes (Figs 504–508). No clinical evidence of their presence may have shown itself till the onset of pain or of deformity and dysfunction from a pathological fracture or till pressure on

or invasion of nerves, has taken place. In a case of spontaneous fracture, therefore, a careful search must be made for any local radiographic evidence of osteoporosis or of bone destruction in the region of the fracture (Fig 509) and for any translucent areas elsewhere in the same or other bones. The finding of an existing primary growth in the breast, kidney, or elsewhere will further strengthen one's suspicions, as also will the history of a primary growth which has existed previously, whether treated at the time with apparent success or not. Even if no present or past primary growth has been found the conviction of the radiologist should not be shaken, for many such primary growths offer no clinical evidence of their presence.

The osteolytic metastasis reveals itself as a ragged translucent area in the bone with similar possibly smaller areas elsewhere. Sometimes these areas become confluent. These metastases start as a rule in the medulla and as they grow invade the cortex from within. Though usually homogeneous the translucent areas may show a mottled appearance and the rest of the bone may show an osteoporotic appearance with multiple small translucent areas. The cortex as a rule shows no expansion till almost complete cortical invasion from within has occurred. Occasionally a slight thickening of the cortex above and below the metastasis is seen rendering the differential diagnosis from a sarcoma difficult in the case of solitary lesions.

Hypernephromatous secondary deposits differ from other metastases in

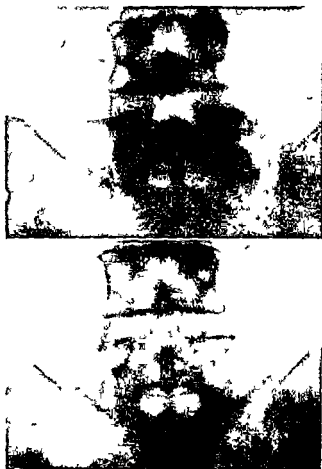


FIG. 506.—Metastasis from carcinoma mammae in transverse process of 10th lumbar vertebra. Radiographed because of severe backache. No bone change shown in first radiogram but clearly shown (destruction of transverse process) in repeat radiogram three and a half months later.

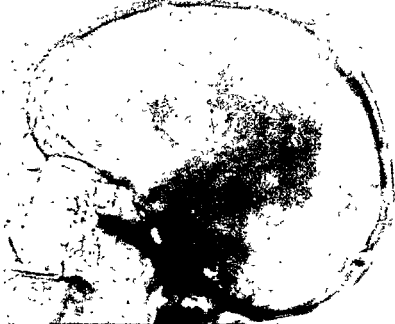


FIG. 507.—Metastatic carcinoma of skull (primary in breast) showing diploic origin of metastases.

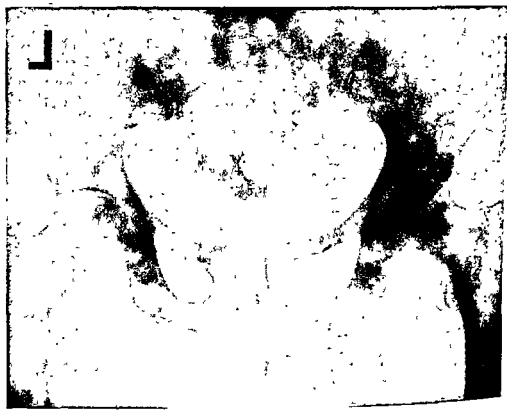


FIG. 508.—Multiple osteolytic metastases from breast carcinoma operated on three years previously

that they show a greater tendency to occur as single lesions. They also more frequently give rise to a bulging of the cortex and to invasion of the soft tissues unlike other metastatic tumours.

In osteolytic metastases in the vertebral bodies translucent areas are seen in the medulla usually in more than one vertebra possibly some distance apart. At first no collapse takes place but as the metastasis increases in size collapse of the body occurs. The intervertebral disc however is not involved (Fig 510).

**OSTEOBLASTIC OR SCLEROSING METASTASES**  
—These arise as a rule from a primary growth in the prostate but may on occasion be found in certain cases of scirrhous carcinoma of the breast (Fig 511). In the former the bone change is usually confined to the pelvis and lumbo-sacral spine but may also involve the upper half of the femur. The characteristic finding in such cases is a general sclerosis or increased density of the affected bones. This increased density is patchy and is often asso-

ciated with small areas of translucency indicating osteolysis combined with sclerosis. The bone may or may not be thickened. Not in-

frequently the discovery of the primary lesion in the prostate follows on the radiographic demonstration of such metastatic bone changes in the pelvis or spine the patient having been radiographed for persistent backache.

### OSTEOGENIC SARCOMA

**Introduction**—At the outset a clear definition of the term osteogenic as



FIG 510—Collapse of a vertebra from metastatic carcinoma. Discs intact.



FIG 509—Metastasis in humerus from breast carcinoma. Only slight X-ray evidence of the metastasis is present yet a pathological fracture has occurred.



FIG. 311 Metastatic carcinomata of pelvis femora etc. Mixed osteolytic and sclerosing type. Primary in breast.

applied to sarcomata seems desirable. *Hertler* defines the term osteogenic as meaning produced by or producing bone. He quotes *Codman* as saying: "These are tumours which are believed to be derived from cells which are supposed to be the common ancestors of the cells which form bone cartilage the fibrous network of bone and the tissue formerly called myxomatous (which from the point of view of pathology is merely a phase of cartilage or fibrous tissue)." In view of this it is not surprising that the histology of bone sarcomata gives rise to considerable confusion of thought and to frequent difficulties in exact pathological interpretation. In their early diagnosis the radiologist must be prepared to face a grave responsibility for unless the diagnosis can be made early it is often useless for the preservation of the life of the patient. In case of bone pains in young patients repeated X-ray examination is frequently of the greatest value. The intervals should not be more than one week and the earliest suspicion of sarcomatous changes must be diligently sought for. To wait till such appearances are sufficiently well developed to be absolutely diagnostic is often fatal. The closest collaboration between the radiologist and the clinician must be maintained and there should be no hes-

tation in soliciting the aid of the surgeon and of the pathologist in the performance of a biopsy. Great as are the difficulties of the pathologist his task can be made hopeless if the surgeon fails to produce for his examination a portion of the tumour which is characteristic.

An excellent account of the responsibilities of the pathologist with special reference to biopsy is that of Young. He regards radiology and biopsy as being more precise as diagnostic procedures than clinical surgery. In cases of radiological doubt he strongly advocates biopsy rather than serial radiography because of the delay entailed with the latter procedure. The method he favours is a decompression of the growth by open operation during which a piece of tissue large enough to be regarded as a fair sample of the lesion should be excised carefully for the purposes of microscopic diagnosis. If the pathological report is not conclusive another piece of tissue should be excised as before with the least possible delay. If the lesion is proved to be a bone sarcoma amputation or wide resection should be carried out immediately. If happily the lesion is not bone sarcoma other appropriate measures should be applied.

Ogilvie on the other hand believes that an opinion based on a careful consideration of the history physical signs radiographic appearances and reaction to radiation is more likely to be right than one derived from the examination of a piece of tissue removed for section. He suggests that we form our decision on such grounds alone.

**Age Incidence**—Most bone sarcomata occur before the age of 20 years but they may on occasion be found at a later age period especially where they occur as a complication of elderly diseases such as Paget's disease (osteitis deformans).

These tumours are commoner in males than in females.

**Sites**—The vast majority of bone sarcomata occur in the lower limb especially in the lower end of the femur, other fairly common sites are the upper end of the tibia and upper end of the humerus. These three sites between them account for two thirds of all osteogenic sarcomata. Rarer sites are the humerus fibula radius ulna and occasionally the scapula. They are never found in the metacarpals metatarsals or phalanges of the hand or foot. They do not traverse cartilage though the author has seen one case in which the upper end of the tibia was involved from extension of a sarcoma of the lower end of the femur presumably via the crucial ligaments.

Pre-existing simple lesions such as osteochondromata (exostoses) chondromata giant cell tumours or Paget's disease may develop sarcomatous changes.

**Clinical Features**—The usual sequence of clinical events is in the following order (1) trauma (2) pain (3) tumour (4) circlexia (5) pathological fracture.

**TRAUMA**—Though bone sarcomata may develop without any clear history of preceding trauma such a history is so frequently obtainable that trauma must

undoubtedly be regarded as a definite localising factor, possibly in a patient with a predisposition to this type of tumour

**PAIN**—The pain, at first slight and intermittent but later becoming more severe and constant, and often worse in the warmth of bed, is due to pressure on, or invasion of, the periosteum. It is usually the earliest symptom (except in the periosteal type)

**TUMOUR**—This usually follows the period of pain, and at first may not be discernible. Sooner or later, however, an indefinite spindle shaped tumour can be palpated if affecting a long bone, or an indefinite swelling if affecting another bone. In the periosteal sarcoma the tumour may be the first clinical sign.

**CACHEXIA**—This is a late occurrence, and is attributable as a rule to metastatic deposits in the lungs, which may on occasion be present before the tumour has reached any appreciable size.

**PATHOLOGICAL FRACTURE**—This is a rarer finding than in metastatic carcinoma, and occurs only when the disease is advanced. According to *Platt* it occurs only in 8 per cent of cases of bone sarcoma (as opposed to 33 per cent occurring in metastatic carcinoma).

### Pathology

**MACROSCOPIC APPEARANCES**—In his account of the pathology of sarcoma *Hertzer* emphasises two points, viz (1) that the surgeon at biopsy should fully appreciate the macroscopic appearances of sarcoma, and (2) that he should select for histological examination, characteristic tumour tissue. One of course assumes that before such a procedure is attempted due regard will have been paid by all concerned to the clinical features of the case, and to its radiographic appearances.

The essential macroscopic findings in bone sarcoma are simultaneous bone proliferation and bone destruction. According to *Kolodny* bone destruction is always in excess of what might be expected from a study of the radiograms. As opposed to osteomyelitis the bone, when cut is more granular and fragile in sarcoma and the tissue is more vascular. In all sarcomata medulla, cortex and periosteum are involved. In rapidly growing sarcomata the periosteal changes may predominate. There may be *mothting* of the cut bone from varying degrees of rapidity of growth, there may be hæmorrhages, there may be cystic changes, there may be invasion of the medulla by new bone formation in solid bone tumours, and there may be secondary tumour formation in the bone, at some distance from the primary growth.

**MICROSCOPIC APPEARANCES**—In the histological examination of bone tumours the chief aim of the pathologist is to differentiate between benignity and malignancy, a task which is often difficult. In sarcomata the characteristic feature of the cell structure is its variability. The more uniform the cell structure the less likely is the specimen to be sarcomatous. A characteristic



feature of the cells of osteogenic sarcoma is the deeply staining nature of their nuclei. In the excessively malignant tumours large, deeply staining mononuclear giant cells are found. In less malignant tumours the characteristic cells may be spindle-shaped, or round, deeply staining cells, with fibrous tissue interspersed. In addition, strands of bone and islands of cartilage may be found. It must be appreciated that deeply staining multinuclear cells are not characteristic of bone sarcoma, but are also to be found in callus tissue and in the region of subperiosteal hæmatomata (myositis ossificans traumatica). They may, however, be found in bone sarcomata, especially if there is degeneration of blood-clot.

**Classification.**—Of the many classifications of bone sarcomata which have been evolved, that of the Bone Registry of the American College of Surgeons, as modified by Ewing, is very helpful. It lends itself readily to a comparison of various types of sarcoma with benign lesions, with which they may be confused radiologically.

# 1. CLASSIFICATION OF THE BONE REGISTRY OF THE AMERICAN COLLEGE OF SURGEONS

## *Types of Sarcoma*

- (a) Periosteal.
- (b) Medullary and subperiosteal.
- (c) Telangiectatic.
- (d) Sclerosing.
- (e) Fibrosarcoma.
- (f) Atypical unclassified.

# 2. EWING'S CLASSIFICATION

## *Types of Sarcoma*

1. Medullary and subperiosteal.
2. Telangiectatic.
3. Sclerosing.
4. Periosteal.
5. Fibrosarcoma. (Medullary ; Periosteal.)
6. Parosteal (including Capsular).

## *Lesions Causing Confusion*

1. Chronic osteomyelitis, etc.
2. Vascular giant-cell tumour.
3. Benign bone hypertrophies.
4. Traumatic and syphilitic periostitis.
5. Extra-osseous lesions.
6. Benign joint affections.

# RADIOLOGICAL APPEARANCES OF OSTEOGENIC SARCOMA

The classification of an osteogenic sarcoma in any particular group is often impossible from the purely radiological point of view. Such a segregation is usually possible only after a careful evaluation of all the various radiological, clinical, and pathological features presented. The degree of vascularity, for instance, can be assessed only by clinical and pathological examination, and is not a feature which lends itself to radiological interpretation.

Osteogenic sarcomata whether medullary, subperiosteal, telangiectatic, or sclerosing, always contain an admixture of osteolytic and sclerosing elements, and all involve, sooner or later, and to a greater or less degree, medulla, cortex and periosteum. One element may predominate, so that in the radiogram the major element may appear as an osteolytic or osteoblastic process. It must be appreciated that the osteoblastic or sclerosing process should be regarded not so much as the sarcomatous disease itself, as the effect of the disease—Nature's evidence of resentment to the disease, and her attempt to limit it. Even the most osteoblastic type of sarcoma contains osteolytic elements though in the radiograms these may be obscured, or to the naked eye they may be apparently obliterated by the sclerosis which is predominantly demonstrated. Another point which must not be overlooked is that a radiogram penetrates the whole substance of the bone so that cortical, periosteal, or even soft tissue changes may obscure, or may be mistaken for, the purely medullary changes, with a resultant tendency to false interpretation of the true state of affairs.

The radiologist should therefore, be content with deciding, if he can, whether the tumour in question is an osteogenic sarcoma or not, leaving refinements of classification to the clinician the surgeon, and the pathologist. No attempt is made in this article to make a radiological differentiation between the various types of osteogenic sarcomata all that can be done is to indicate the various types of bone change which may be encountered in such a tumour. Even so too much emphasis cannot be laid on the necessity for the careful investigation of the history of the case and for the clinical examination, along with the radiological findings. With these data before him the radiologist is often in a position to offer a firm diagnostic opinion. To make a diagnosis on radiological evidence alone is sometimes justifiable, but to do so regularly is to court disaster. In many cases even with all the above mentioned data available an element of doubt may still be present, in such cases no hesitation should be shown in calling in the aid of the pathologist (provided that the surgeon undertaking the exploratory operation has a full appreciation of the macroscopic appearances presented by osteogenic sarcomata and is competent to provide the pathologist with characteristic tumour tissue for investigation). The responsibilities of all concerned are so great that every member of the team should fully appreciate his limitations. It is equally culpable to remove a limb, in part or in whole in the case of an innocent tumour, as to fail to do so in the case of a malignant tumour where such a procedure might have resulted in the saving or even the prolongation, of a life.

Returning however to the responsibilities of the radiologist, we enquire what radiographic appearances should arouse in his mind the suspicion of osteogenic sarcoma (due regard having been paid to the history of the case and to the results of clinical examination). They are as follows (but all are not necessarily present at one and the same time)

1 Bone Destruction—The first sign may be a localised osteoporosis but soon irregular areas of translucency single or multiple may be seen in the medulla or cortex or in both (Fig. 512). These may be confluent and they may be localised or extensive. Wherever the tumour has originated sooner or later both cortex and medulla are involved. The line of least resistance is met with in the medulla so that extensive medullary destruction may be the predominant feature. Where the cortex is involved the tumour may extend beyond it, leaving remnants of the original cortex remaining *in situ* (in contradistinction to Ewing's tumour where the cortical remnants may be pushed out). The outlines of the areas of destruction are blurred from invasion. Extension of destruction through cartilage is rare because of its relative avascularity so that the destructive area rarely extends beyond the epiphyseal cartilage (Fig. 513) before fusion of epiphysis with diaphysis (except by extension from without) or beyond articular cartilage in older patients where such fusion has taken place. In early cases the only sign visible on the radiogram may be a slight cortical erosion of the bone and when such an appearance is accompanied



FIG. 1. Pathological fracture of femur in osteogenic sarcoma.



FIG. 513.—Osteogenic sarcoma of femur showing lifting of epiphysis rather than invasion.

by a painful tumour the possibility of an early osteogenic sarcoma must be seriously considered. Confirmation may be sought either by a limited number of repeat radiograms or by biopsy.

## 2 Subperiosteal New Bone Formation

(a) LONGITUDINAL LAYERS—In showing their reaction to the invasion of the tumour cells the subperiosteal osteoblasts may form layers of new bone beneath the raised periosteum (Figs 514-518).

This formation of a thin layer or layers of new bone parallel to the shaft under the raised but intact periosteum may be one of the earliest radiographic signs of an osteogenic sarcoma. From



FIG 514—Osteogenic sarcoma showing on peel layers and radiating spicules



FIG 515—Osteogenic sarcoma showing sun ray spicules and reactive triangle

the X ray point of view it is however difficult to differentiate this from a simple traumatic or syphilitic periostitis. The subsequent progress of the case the absence of similar lesions elsewhere and the Wassermann reaction should however help in the differentiation.

#### (b) SUNRAY SPICULES

—As the periosteum is being raised new bone may be laid down beneath it in columns at right angles to the surface of the bone presenting characteristic perpendicular parallel or radiating spicules in the radiogram (Figs 14-16). Though often regarded as characteristic it must be remembered that such spicules occur in only about 18 per cent of osteogenic sarcomata (Kolodny) and that similar spicules may be found in certain cases of chronic osteomyelitis and in syphilitic periostitis.

After the growth has penetrated the periosteum strands of new bone may be formed but they now lose their columnar arrangement and grow in all directions.

#### (c) REACTIVE TRIANGLE +

##### OR SUBPERIOSTEAL CUFF —

At the periphery of the tumour the subperiosteal osteoblasts are still fighting a losing battle but they still show their reaction by forming a barrier of new bone. In the radiograms this is often demonstrable by a cuff of new bone (Fig 518) shown as a triangle of subperiosteal new bone merging into the surface of the bone above and below the tumour and with an ill-defined edge. Between these limits the subperiosteal new bone has been destroyed by the advancing tumour so that in this intermediate part no evidence of it is visible in the radiogram.

3 Mixed Nature of X ray Changes — An osteogenic sarcoma is either wholly



FIG 518 — (a) Sarcoma of humerus. X ray appearances resembling osteomyelitis. No pus found at operation. (b) The same case two months later. Now obvious diagnosis of osteogenic sarcoma confirmed at operation.

osteolytic or wholly osteoblastic. In all cases there is evidence both of bone destruction and of new bone formation. According to the type of tumour however one or other of these processes may predominate.

4 Soft tissue Tumour—Sooner or later the invading cells perforate the periosteum and the tumour extends into the soft tissues. Such a soft tissue tumour will usually be demonstrable in the radiogram and should always be looked for. Clinically such a tumour will be lacking in free mobility; it will show no signs of inflammation; there will as a rule be little or no impairment of movement in the adjacent joint; it will be of greater size than the underlying X-ray changes warrant and it will show no signs of pedunculation.



FIG. 517—Simple periosteitis with palisade spicules.

### SPECIAL FEATURES OF CERTAIN OSTEOGENIC SARCOMATA

1 Sclerosing Osteogenic Sarcoma—In this tumour the osteoblastic or sclerosing element predominates. This sclerosing process may be seen either in the medullary or in the subperiosteal regions or in both masses of dense ossified tissue being formed and the whole tumour showing a very dense structure in the radiogram (Fig. 519). It may spread up the marrow cavity with the consequent involvement of a considerable area of the shaft in a club-like mass. It may also extend into the epiphysis.

In these cases the reactive triangle is absent and one of the earliest signs may be a condensation of the medullary region of the bone without any apparent cortical or subperiosteal involvement. In some cases the subperiosteal change may merely be shown as a roughening of the surface of the bone (resembling osteomyelitis).

2 Telangiectatic Osteogenic Sarcoma—All sarcomata are vascular and the

production of a so-called "malignant bone aneurysm" is merely a matter of degree. In extreme cases sacs or cysts are formed, filled with blood, or there may be a series of communicating blood sinuses lined by tumour tissue. In such cases expansile pulsation will be demonstrable (Fig. 520).

They occur most commonly in children, they are characterised by rapid bony destruction and are very liable to pathological fractures. The periosteum is perforated at an early stage, they grow rapidly, metastasise early and are usually fatal within a few months.

Because of the extreme degree of bony destruction and of the formation of blood sinuses, the main radiographic features are essentially those of marked osteolytic changes. New bone formation may be very slight or even absent.

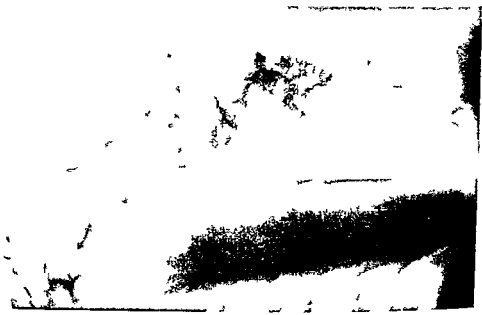
3. **Periosteal or Extraperiosteal Osteogenic Sarcoma**—This type of sarcoma originates from the outer layers of the periosteum and envelops rather than involves the bone. In such cases the bone itself may, therefore, show no appreciable change or it may be merely superficially eroded (from pressure



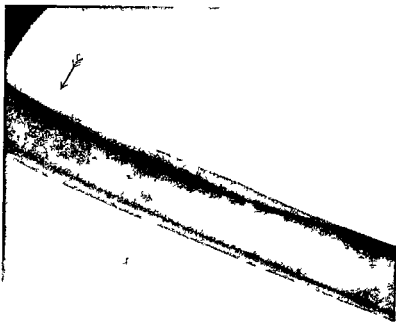
FIG. 518.—Early osteogenic sarcoma with local osteoporosis and well marked reactive triangle.



FIG. 519.—Sclerosing osteogenic sarcoma of humerus.



110 5 0-11  
 a c a 1 1 1  
 b, cal, fra  
 soft wood  
 x et lo K t o r) 1 atiol  
 y m (7 tel k et o at age o  
 y a i l h lo O t m!



1 G B 1-1 row o f cortex of fen r from ext a  
 p loste 1 w o l c l m later ext a saron  
 l hat us net a tes were f l along l l ally 1 1



rather than from invasion) Occasionally irregular calcified extraperiosteal strands may be seen in the tumour, without any sunray arrangement

They are commonest in the femur and may occur in the midshaft

These tumours are very malignant and show early metastasis in the lungs or sometimes in the brain The lung metastases may in some cases be demonstrable before the primary tumour has been diagnosed (Fig 521)

4 **Fibrosarcoma (Medullary and Periosteal)**—These are slow growing tumours which arise in the marrow cavity or on the surface of the periosteum They are relatively benign and may not recur after excision

The medullary type originates in the metaphyseal region, it causes expansion and destruction of the diaphysis but does not involve the epiphysis it may perforate the periosteum and extend into the soft tissues the cortex being eroded later from pressure Radiologically it presents an appearance like that of a solid opaque giant cell tumour with no septa

The essential difference between these tumours and malignant osteogenic tumours is that they show a sharply defined limit as opposed to the ragged blurred limits of the latter.

In the periosteal type there is X ray evidence of solid and opaque masses on the surface of the bone (periosteal) causing no bone change in the shaft, and displacing rather than invading the soft tissues

These tumours are relatively benign hence the importance of differentiating them radiologically from the malignant osteogenic sarcoma



FIG 522.—Sarcoma of lower end of femur superimposed on Paget's disease

3. **Capsular and Parosteal Sarcoma** —These tumours may be derived from (a) the synovial membrane (b) the joint capsule or (c) the bone near a joint.

The first two types which are rare may show no bone change or at the most a slight general rarefaction. The third type may show  $\lambda$  ray appearances which are radiologically indistinguishable from those of tuberculosis.

### SARCOMA SUPERIMPOSED ON PRE EXISTING DISEASE

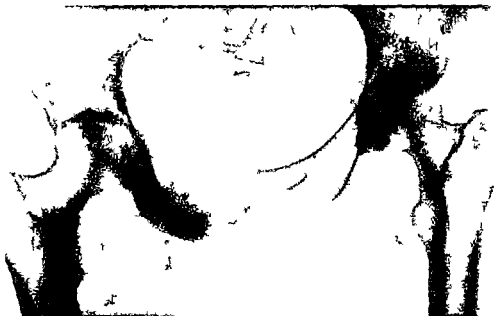
**Chondroma Osteochondroma** —Any tumour composed in part or in whole of cartilage may undergo sarcomatous changes. Where a sudden rapid



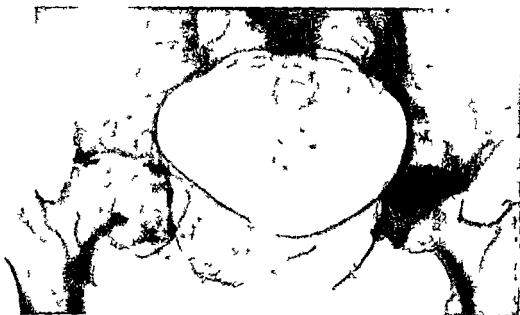
FIG. 523. —Multiple osteogenic sarcomata of skull superimposed on Paget's disease (confirmed by autopsy). Sarcomata were also found in the pelvis and humeri.

growth in such a tumour occurs the possibility of supervening malignant disease must be seriously considered. In the radiogram one or more of the bone changes already described under osteogenic sarcoma will be shown.

**Paget's Disease (Osteitis Deformans)** —The bone changes characteristic of this disease have been described elsewhere in this book. Superimposed tumours either osteomata or osteogenic sarcomata may occur. The incidence of the latter is in about 7 per cent. of all cases of Paget's disease. Where in a



(a)



(b)

FIG. 1. (a) Osteogenic sarcoma of pubic ramus before X-ray treatment. (b) The same after prolonged X-ray treatment. Without the previous history the radiographic appearances in (b) would simulate those of a giant-cell tumour.

case of Paget's disease there arises marked localised pain or evidence of nerve pressure other than that normally occurring due to bony thickening at the cranial or other foramina radiographic evidence of osteogenic tumour formations should be sought. The X ray appearances of such resemble those already described under medullary or subperiosteal osteogenic sarcoma (Figs 522, 523). They are however less characteristic. Sunray spicules are as a rule absent the X ray appearances being those of irregular destruction with usually some remnants of the old bone within the tumour. Occasionally some new bone formation can be made out.

### MASKING OF X RAY APPEARANCES OF OSTEOGENIC SARCOMA BY PREVIOUS IRRADIATION

When treated by X rays especially if such treatment has been intensive or prolonged calcification and ossification take place in and about the tumour. In such cases the typical radiographic appearances of the tumour are altered. If a case has been treated in this manner unless the history of such irradiation is volunteered or obtained and due allowance made for it an error of diagnosis is possible (Fig. 524).

### EWING'S TUMOUR (*syn* DIFFUSE ENDOTHELIOMA OF BONE ENDOTHELIAL MYELOMA)

**Age Incidence**—This is essentially a tumour of childhood and adolescence. The majority of cases occur before the age of 30 years, relatively few being seen after this age though this may occur. Males are more prone to it than females (*Kolodny*).

**Sites**—The distribution of this tumour differs somewhat from that of osteogenic sarcoma in that it more commonly affects the small bones (such as the os calcis) and the flat bones (such as the innominate bone). The commonest sites are the midshafts of the femur, the tibia and the humerus. It may occur in other bones.

Though it sometimes originates in the end of the long bones it rapidly extends along them so as to soon involve a large part of the shaft. In the early stages the epiphysis is not affected.

**Clinical Course**—In osteogenic sarcoma the patient prior to the development of the tumour is usually in good health. In Ewing's tumour on the contrary patients are often of a delicate constitution and many of them give a history of nutritional disorders in childhood or of a recent attack of focal sepsis. The unaffected bones may show evidence of osteitis fibrosa (*Ewing*). A history of preceding trauma is often obtainable.

The onset is slow, the patient complaining of pain in one bone. This may be merely a dull ache associated with cramps or it may be of a sharp shooting character. At first intermittent it becomes constant and severe. The pain is frequently associated with intermittent attacks of fever.

Sooner or later a tumour develops, but this does not necessarily show a progressive increase in size, it may temporarily subside or it may grow rapidly. In its early stages the condition may be confused clinically with osteomyelitis, being often accompanied by fever and leucocytosis. Later, however, the tumour formation, with occasionally a spontaneous fracture, points to the more serious nature of the lesion. Spontaneous fracture may, in some cases, occur comparatively early in the disease.

At first the bony tumour is a single lesion, later, however, tumours in other bones such as the skull, pelvis, vertebræ, ribs, or long bones, are formed by metastasis. Metastases may also appear in the lungs, though usually at a relatively later period than in osteogenic sarcoma.

The disease is almost invariably fatal, death occurring within a few months to a few years, in spite of recession of the primary tumour as a result of radiotherapeutic or surgical treatment, the prognosis is not that of the primary tumour, but that of the metastases.

**Pathology.**—As a result of microscopic examination Ewing regards this tumour as originating in the perivascular endothelium of the bone or bone marrow. The cells are not definitely spindle shaped, but occur in sheets of small, deeply staining polyhedral cells with no intercellular material, and they may form capillary blood vessels or blood spaces containing unaltered blood. As the cells multiply the Haversian canals are expanded. The shaft shows a widening with bone absorption, the soft tissues eventually being invaded.

Metastases occur in the skull, the ribs, pelvis, and other bones. Eventually the whole marrow system may be involved (the distribution then resembling that of myelomatosis), hence arises the necessity for radiographing the whole skeleton, especially the skull. On the other hand, the lesion may persist as a solitary tumour, with recovery under adequate radiotherapeutic or surgical treatment.

Lung metastases are comparatively late in their appearance. The lymphatic glands may be enlarged as a result of dissemination via the lymphatics.

A striking feature of Ewing's tumour is its radio sensitivity. As small a dose as 1,000r may suffice to bring about complete recession. Unfortunately, however, metastases in other bones may have taken place before the X ray treatment of the primary tumour has been initiated. It is disappointing to produce apparent cure in the primary tumour, only to find evidence of metastases elsewhere. The metastases are equally responsive to X ray treatment, but once widespread metastases have occurred, the task of following them up with X ray treatment is hopeless. However, on the off chance that the tumour found may be a solitary one, and that metastases may not occur, it is worth while treating it radiotherapeutically.

Such treatment is sometimes employed as a diagnostic test, in differentiating Ewing's tumour from osteogenic sarcoma (which is more radio resistant). Stewart Harrison states that "a tumour that does not disappear by X ray

treatment is not a Ewing's sarcoma. He prefers test irradiation to microscopic examination as a diagnostic test.

**Radiological Appearances**—The X-ray appearances of this tumour are by no means characteristic. As a rule the history and clinical features of the case demand the closest attention in conjunction with the X-ray appearances presented before a diagnosis of Ewing's tumour may be put forward.

The bone changes are seen as a rule to involve extensively the mid-shaft rather than the metaphysis (Fig 595). In the early stages they closely resemble those of osteomyelitis. They consist of an endosteal and subperiosteal fusiform thickening of the bone gradually fading at each end. The subperiosteal new bone may be in the form of onion peel layers which are regarded by some as characteristic. Such appearances may however be misleading as precisely similar appearances may be found in syphilitic periostitis. Occasionally parallel or radiating spicules may be seen in this tumour but again similar appearances may be seen in osteogenic sarcoma in syphilitic periostitis and more rarely in lymphadenomatous involvement of bone.

These subperiosteal and endosteal changes are accompanied or more often followed by irregular destructive changes in the cortex and medulla resembling those of osteomyelitis. *Herendeen* summarises these changes as (1) a reduction in opacity (2) a loss of structure (3) an increase in breadth of the spongiosa (4) a thinning of the cortex. He describes



FIG 595—Ewing's tumour of tibia confirmed by biopsy

the destructive changes as often resembling an erasure of the bony shadows in the radiogram by means of an indiarubber.

As a result of the destructive bony changes pathological fracture may occur. No infrequently as a result of the X-ray findings and of the history and

clinical features of the case the surgeon cuts down on to the bone regarding the case as one of osteomyelitis and expecting to find pus. Where such is not found the possibility of Ewing's sarcoma should be immediately considered and further investigation should be made either by a test irradiation or by a microscopic examination of a suitable portion of tissue.

The X ray diagnosis of Ewing's tumour is often one of extreme difficulty many cases are diagnosed as Ewing's tumour which are not this lesion and many cases of Ewing's tumour are probably missed through an inadequate correlation of the X ray findings with the history and clinical features of the case.

### OTHER VARIETIES OF ENDOTHELIOMA OF BONE

**Solitary Bulky Cystic or Telangiectatic Angio endothelioma**—This is a rare destructive single tumour which affects the end or the shaft of a long bone of adults.

It grows steadily perforates the cortex and invades the soft tissues.

It recurs and produces pulmonary metastases.

It may give rise to expansile pulsation a bruit being heard by auscultation. Radiologically and clinically it resembles telangiectatic osteogenic sarcoma it shows no reactive triangle. It grows more rapidly than the aneurysmal type of giant cell tumour.

**Multiple Endothelioma of Bone**—A case of this type was described by *Marekwald*.

Small tumours of the bone marrow were found affecting more particularly the vertebrae sternum skull and pelvis.

Radiologically it cannot be differentiated from myelomatosis the differential diagnosis being essentially a histological one.

### SCHULLER'S DISEASE (A RETICULO ENDOTHELIOSIS)

*Morison* gives a complete account of this disease which is associated with the production of multiple tumours in the bones especially the skull pelvis and more rarely the long bones. These tumours are of the nature of a hyperplasia of the reticulo endothelial cells. Cases of this disease have been recorded under a variety of titles including the Hand Schuller Christian syndrome Xanthomatosis Lipoid Granulomatosis and other variations. The generic term Xanthomatosis was coined by *Roulin* to include all cases in which a disturbance of lipid metabolism occurs such as the Hand Schuller Christian syndrome Gaucher's disease Niemann Pick's disease and the various forms of Xanthoma Xanthomyeloma and Xanthelasma.

**Clinical Features**—According to the localisation of the lipid deposits exophthalmos (retro ocular deposits) or diabetes insipidus (pituitary location) may be present. Other signs depending on the nature of the lipid deposited in the cells that may be shown are fat above the clavicles and about

the genitalia (dystrophia adiposis genitalia) spongy gums (when the deposit is periodental) enlargement of the spleen liver and lymphatic glands discharging ears brown patches on the skin at post mortem yellow spots on the pleura peritoneum dura and periosteum may be found

**Radiological Appearances**—Radiologically the characteristic finding is the presence of multiple sharply defined map like erosions in the cranial vault with possibly areas of destruction in the pelvis femora and other bones sometimes accompanied by pathological fractures These destructive areas are associated with soft tumours The cranial sutures may show gaping from increased intracranial pressure

All the tumours respond to treatment by X rays This condition is considered in detail on pages 537-540 of this volume

### MYELOMA (MULTIPLE MYELOMATOSIS)

**Age Incidence**—This is a disease of adult life usually found after the age of 50 years and affecting males more frequently than females

**Sites**—The distribution is usually widespread Any bone may be affected especially the ribs pelvis spine femur sternum scapula humerus skull and clavicle

**Clinical Features**—The earliest symptom is usually pain this may be slight and indefinite but is often very severe Occasionally pathological fracture is the first sign of the disease Deformities from bone expansion or from destruction and soft tissue invasion may be found

**Pathology**—These tumours are usually multiple each tumour being essentially a primary one They are therefore due to a systemic disease rather than true tumours

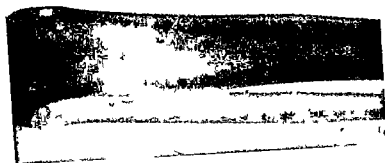
They are derived from the specific bone marrow cells viz granular myelocytes lymphocytes and nucleated red blood cells The commonest form of myeloma according to *Ewing* is composed of plasma cells whose origin is doubtful—possibly from lymphocytes or from endothelial cells According to the type of cell from which they are derived myelomata are therefore divided into four histological groups viz (1) plasma cell tumours (2) lymphocytomata (3) myelocytomata (4) erythroblastomata

They have certain common features which differentiate them from osteogenic sarcomata They are usually multiple but may occur as solitary tumours They are entirely osteolytic and never give rise to new bone formation They do not tend to produce lung metastases Neighbouring lymphatic glands may be enlarged They are found in the mid portion rather than the ends of bones and frequently occur in the ribs vertebrae and skull—unusual sites for osteogenic sarcoma By rapid destruction of the bone they invade the soft tissues and are often the site of pathological fractures Bence Jones proteoses are found in the urine in about 50 per cent of the cases The patient becomes anæmic and cachectic Unless existing as a solitary tumour which is rare the

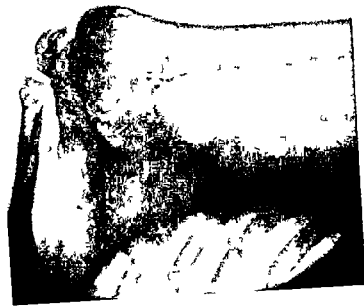




(a)



(b)



(c)

FIG 530 —Multiple myelomatous Note the clear cut outlines of the translucent areas

prognosis is bad death occurring in anything up to two to three years. These tumours are radio sensitive.

**Radiological Appearances**—The first X ray evidence of the disease may be a mottling of the affected bones. Later this gives place to multiple punched out areas of destruction of varying sizes discrete or confluent and with clearly defined borders (Fig 526). Pathological fractures especially of the ribs are not infrequent and deformities from bony destruction are common.

There is a complete absence of new bone production, this is a distinctive radiographic feature of the disease. In the spine there may be destruction of the vertebral bodies together with destruction of the discs (the latter feature serving to differentiate it from metastatic carcinoma in which the discs are intact). Collapse of the vertebral bodies occurs at an earlier stage than is seen in metastatic carcinoma with the production of kyphotic deformities. In the skull punched out areas of destruction are seen which may be difficult to differentiate from those found in metastatic carcinoma. The pelvis may show diffuse areas of destruction giving the bone a bloated or puffy appearance.

## RARER TUMOURS OF BONE

### Chloroma

**Age Incidence**—This is a disease of early life only being found occasionally in adults.

**Sites**—The usual site is the skull but occasionally the spine ribs sternum pelvis and long bones are affected.

**Clinical Features**—Clinically the disease resembles an acute and rapid type of leukaemia either myelogenous or lymphatic. When the skull is involved the symptoms are referable to the eye ears nose or throat.

It is invariably fatal.

**Pathology**—As its name implies this is a greenish coloured tumour. It is allied to the leukaemias and may be found either as a myelogenous or as a lymphatic type.

**Radiological Appearances**—*Geschickter* and *Copeland* describe a case of a male aged 17 years whose skull and humerus were involved. The radiogram of the humerus shows destructive changes with a pathological fracture. Subperiosteal new bone formation with a slight palisade appearance but no reactive triangle is shown. Beyond the tumour punched-out areas are seen in the cortex. The skull is recorded as showing bony destruction.

### Lymphoma

In a small percentage of cases of Hodgkin's disease (lymphaderoma) bony involvement may occur and may give radiographic evidence.

**Age Incidence**—The condition occurs usually in adult life.

**Sites**—The lesion is usually found in the vertebrae, the long bones, skull sternum, and ribs are less frequently involved

**Clinical Features**—The clinical features are those of Hodgkin's disease the bony involvement, heralded by pain, occurring a few years after the disease has become manifest

**Pathology**—There is an involvement of the bone marrow in the lymphadenomatous process with fibrosis

**Radiological Appearances**—The bone changes may be of the nature of a subperiosteal bone proliferation, with sometimes the production of palisade spicules. Or it may be of the nature of a bone destruction

In the vertebrae the changes may take the form of an increased density of one or more vertebral bodies, or it may show as a purely destructive process with collapse

Sometimes the proliferative and destructive changes may co exist in one bone

### Myosarcoma and Lipoma

These are rare types of tumour in bone, which reveal themselves as destructive areas of the nature of recurrence in the vicinity of the site of removal of such tumours of the soft tissues. Presumably the bone was slightly involved at the time of operation. Death may take place from pulmonary metastases

### Neurogenic Sarcoma of Bone

Tumours derived from nerve tissue may be benign or malignant. The malignant type resembles fibrosarcoma

**Age Incidence**—They occur as a rule after the age of 30 years

**Sites**—They are usually found in the long bones, but occasionally affect the vertebrae, as the malignant variety of the "hour glass" tumour

**Pathology**—Softer than the fibrosarcomata, these tumours may present either a beefy red or a



FIG 574.—Hour glass tumour of spine showing erosion of pedicles and of posterior surfaces of vertebral bodies. In the antero posterior view an oval paravertebral soft tissue shadow could be made out

grey gelatinous appearance. They may involve not only the deep seated nerve trunk but also the bone and the neighbouring joint.

**Radiological Appearances**—The bone may be eroded from without or it may show destructive areas sometimes extensive and accompanied by pathological fracture. The periosteum may be raised and subperiosteal new bone may be shown. A soft tissue swelling is usually shown and sometimes the adjacent joint is invaded. In spinal hour glass tumours the characteristic feature is erosion or even disappearance of the pedicle with possibly an erosion of the posterior surface of the vertebral body (Fig. 527) and the demonstration of an oval or rounded soft tissue tumour in the paravertebral region. This is of course most readily shown in the thoracic region when its shadow is well shown against the background of translucent lung tissue. It can however be demonstrated in the cervical and lumbar regions.

## SUMMARY OF BONE CHANGES WHICH MAY BE SEEN IN RADIOGRAMS OF A BONE TUMOUR

### Changes of Structure

- (a) No change (extraperiosteal sarcoma)
- (b) Increased density
  - (i) Due to thickening
  - (ii) Due to increased calcium content
    - Ischæmic (paravertebral or spinal tumour)
    - Sclerosing metastatic carcinoma
    - Vertebral lymphoma
  - (iii) Due to both thickening and increased calcium content
    - (Sclerosing osteogenic sarcoma sarcoma after X irradiation)
- (c) Increased translucency
  - (i) Hyperæmic osteoporosis (parosseous sarcoma)
  - (ii) Destruction and replacement by translucent tissue
    - (a) **HOMOGENEOUS**
      - (i) *With or without trabeculation*
        - Bone cysts
        - Fibrosis of bone
        - Osteoclastoma
        - Chondroma
        - Diaphyseal aclasis
        - Hæmangioma
      - (ii) *Without trabeculation*
        - Osteolytic sarcoma
        - Ewing's tumour
        - Metastatic carcinoma
        - Inflammatory lesions (Brodie's abscess)

**(b) PATCHY**

- Disuse atrophy
- Osteomyelitis
- Sarcoma
- Ewing's tumour
- Metastatic carcinoma
- Myelomatosis (early)

**Changes in Contour****(a) Hyperplastic****Osteoma**

- 1 Simple
- 2 Traumatic (myositis ossificans traumatica)

**(b) Exostotic****Osteochondroma**

- 1 Single exostosis
- 2 Multiple exostoses (diaphyseal aclasis)

**(c) Expansion of cortex**

- Bone cyst
- Fibrosis of bone (osteitis fibrosa cystica)
- Osteoclastoma

**(d) Perforation of Cortex.**

- Osteoclastoma
- Bone cyst with fracture.
- Chondroma
- Metastatic carcinoma
- Sarcoma
- Ewing's tumour
- Inflammatory lesions

**(e) Subperiosteal bone proliferation**

- Simple and traumatic periostitis
- Syphilitic periostitis
- Chronic osteomyelitis
- Subperiosteal calcification and ossification (myositis ossificans traumatica, scurvy)
- Sarcoma
- Ewing's tumour
- Lymphoma

## CHAPTER XLVII

### CYSTS OF BONE

At the outset one is faced by two great difficulties. In the first place the radiologist must appreciate that many conditions may give the radiological appearances of cysts which are not cysts, and in the second place the pathological literature on the subject of bone cysts and fibrocystic disease is to say the least of it somewhat confused.

#### RELATION BETWEEN THE SIMPLE CYST AND THE OSTEOCLASTOMA

The supposed relationship between a simple bone cyst and an osteoclastoma (or benign giant cell tumour) arises from the histological discovery in it of giant cells or osteoclasts. These osteoclasts are however relatively smaller and their nuclei fewer than in the giant cell of the osteoclastoma. The age incidence, the after history and the difference in site of origin are arguments against the common nature of cysts and osteoclastomata. Many authors however maintain that the simple bone cyst may be regarded as a quiescent healing or healed stage of an osteoclastoma. Cysts whose walls contain many giant cells are regarded by them as an intermediate stage and have been designated 'osteoclastomatous cysts'. Osteoclasts however are not significant aetiologically and too much importance should not be attached to their presence. They merely indicate a type of bone absorption. 'Osteoclastomatous cysts' may be regarded merely as simple cysts in a stage of active resorption of bone. While admitting that cysts may be found in benign giant cell tumours, *Elmslie* states that there is not at present sufficient evidence to decide definitely how cysts are actually formed. There seems to be evidence however that they may be formed either as the result of the action of osteoclasts or by degeneration and liquefaction in the fibrous tissue.

#### RELATION OF THE SIMPLE CYST TO FIBROCYSTIC DISEASE

The fibrous connective tissue wall of the cyst and its fluid or solid mass of fibrous tissue (into which hæmorrhage may have taken place) corresponding as it does to the structure of the more widespread lesions of fibrocystic disease has led many to regard the simple bone cyst as merely a local manifestation of fibrocystic disease.

Others regard local fibrocystic disease as being one of "fibrosis" of bone in which cyst formation takes place only to a very slight extent from small

ners of degeneration. One must of course appreciate that the relation between generalised osteitis fibrosa and hyperparathyroidism suggested by *Mandl* in 1926 and conclusively proved by *Donald Hunter* in 1931 has thrown the whole question of fibrocystic disease into the melting pot. Authors prior to that time in describing their cases have probably confused the generalised variety of this disease (hyperparathyroidism) and osteitis deformans (Paget's disease) the local radiogram of these conditions may present similar features but we now know that they can be clearly distinguished pathologically, clinically and biochemically.

The radiographic relationship of the simple cyst and fibrocystic disease is discussed under the heading of Fibrosis of Bone.

### CLASSIFICATION OF BONE CYSTS

In attempting a radiological classification of bone cysts it is necessary that a due regard should be paid to their pathology and clinical features. In this connection the classifications by *Bloodgood* and by *Knaggs* are of interest.

*Bloodgood* in classifying bone cysts groups them as follows:

#### *Bloodgood's Classification*

##### A. True Cysts

- 1 Single cysts without any connective tissue lining
- 2 Cysts with a connective tissue lining which can be peeled off from the bony shell
- 3 Small cyst or cysts in a solid mass of fibrous tissue
- 4 No cyst but a solid mass of fibrous tissue
- Multilocular cysts (fluid or fibrous tissue)

##### B. Cysts in the medullary cavity due to other causes

- 1 Cysts in the cartilaginous tumour and enchondroma
- 2 Pure myxoma with or without cysts
- 3 Cysts in benign giant cell tumour
- 4 Cysts in arthritis deformans and osteitis deformans
- 5 Cysts due to subperiosteal haematoma
- 6 Gullus cysts

*Lawford Knaggs* in classifying the clinical types of osteitis fibrosa divides them into four groups:

- 1 A lesion represented by a uniform mass of fibrous tissue
- 2 A solid mass of fibrous tissue degenerating into one or more cysts
- 3 Cases in which bone is developed and the disease shows signs of coming to an end
- 4 Single cysts of bone

From a correlation of the pathological, clinical and radiological features the following is put forward by the author as a reasonable radiological classification:

## Radiological Classification of Bone Cysts (Roberts)

- 1 Simple cysts
  - (a) Solitary bone cyst
  - (b) Multiple cysts
  - (c) Multilocular cysts
- 2 Fibrosis or fibrous replacement of bone
- 3 Cysts in generalised diseases (such as hyperparathyroidism osteitis deformans osteomalacia Xanthomatosis Gaucher's disease osteogenesis imperfecta etc)
- 4 Myxochondromatous cysts
- 5 Traumatic cysts (e.g. in the carpal scaphoid)
- 6 Infective cysts (e.g. dental)
- 7 Developmental cysts (e.g. dentigerous)
- 8 Arthritic cysts
- 9 Hydatid cysts
- 10 Fibromatous cysts (chronic fibrous osteomyelitis)

## 1 Simple Cysts

Radiologically the solitary bone cyst is as a rule an accidental discovery made during the course of an X ray examination following trauma which though slight may have resulted in a pathological fracture. The area usually affected is the upper end of the humerus femur or tibia the age of the patient is usually under 21 years. The epiphysis of such a patient has not yet united to the diaphysis and the cyst is found in the diaphysis usually near the epiphyseal line it may however be more centrally placed in the diaphysis this altered position being due to the cyst having existed for some years and the bone having grown in length in the meantime.

In the radiogram the cyst is characterised by an area of translucency in the bone surrounded by a smooth thinned cortex which usually shows expansion there may be some trabeculation in the translucent area.

Pathological fractures are frequently found. The subsequent behaviour of the bone thus fractured is sometimes characteristic for the fracture in many cases unites and the bone in this part becomes stronger after the fracture than it was before. The healing process is accelerated if a local store of calcium be introduced either in the form of a paste or of a bone graft after preliminary scraping.

Simple cysts may be multiple or they may become multilocular from fusion of adjacent multiple cysts or from division of a single cyst by septa.

## 2 Fibrosis of Bone

Group 1 of *Knaggs* classification would correspond to the lesion which *Elmslie* later designates fibrosis of bone. Is the difference between this and the solitary cyst merely one of degree? The fibrous tissue in the more extensive lesion is sharply differentiated from bone and radiologically it



presents all the appearances of a large cyst with a central translucent area, a thinned, overlying, cortical expansion—general rather than fusiform—and bending or fracture. The bending is, however, not necessarily the result of fracture, for obvious mechanical reasons the solitary bone cyst, with its adjacent areas of strong healthy bone, will be more likely to break than bend, the converse holding when the greater part of the bone becomes transformed into fibrous tissue. Cysts however, may arise in the fibrous tissue from lique-



FIG 598.—Fibrous replacement of bone

factive degeneration. The difficulty facing the radiologist is that a fibrous mass in the bone and a cyst, either in the fibrous mass or in the bone itself, are of an equal degree of radiotranslucency. It seems at the present time desirable to retain the term 'cyst' for the more localised condition in which local fusiform expansion and pathological fractures are more likely to occur, and to designate the more extensive translucent areas (often seen in more than one bone) in which general rather than local fusiform expansion has occurred, with or without bending or fracture, as "fibrosis of bone" (Fig 528). If, instead of the latter term, one uses the older one of 'fibrocystic disease,' one must clearly realise that the translucent areas are due to fibrous replacement rather than to cyst formation.

### 3 Cysts in Generalised Diseases

Though degenerative cystic areas may occur in any or all of the conditions mentioned in the list above, it must be appreciated that many of the trans-

lucent areas which in the radiogram look like cysts are not really cystic in nature. For instance in osteitis deformans one of the essential features of the disease is resorption of old bone. The affected area is therefore translucent and as the bone may be broadened and may show a thinned but intact cortical layer it looks expanded and so may simulate a cyst. Its subsequent behaviour however does not correspond to that of a cyst: it tends to fill up with coarse trabeculae of new bone. Whilst true cysts may occur in osteitis deformans (probably much less frequently than is generally supposed) it does not by any means follow that every translucent and apparently expanded area in the radiogram of this condition is due to a cyst. *Knaggs* states that he has not met with a single instance in the many bones of Paget's disease which he has examined in which the cystic nature of an intraosseous space was beyond doubt. *Bloodgood* suggested that all such instances as have been recorded may have been really cases of osteitis fibrosa.

The same difficulty of interpretation of translucent areas may occur in any condition in which resorption occurs. The radiological or even the histological differentiation between a local manifestation of this process and of fibrous replacement or of cyst formation may be extremely difficult or impossible. As however the diagnosis of the general disease is of greater importance than the diagnosis of a cyst this feature need not be stressed further. The radiographic examination of the rest of the skeleton, the clinical picture and the biochemical findings will as a rule give sufficient combined information to make the diagnosis clear and the cyst and its differential diagnosis rather fade into the background.

#### 4 Myxochondromatous Cysts

Myxomatous degeneration may take place in a chondroma. From the radiographic point of view it may be very difficult to differentiate between a central or medullary enchondroma, a myxochondromatous cyst and a simple cyst (see chondroma). Fractures are more likely to occur in the simple cyst and the myxochondromatous cyst.

As pure tumours of bone myxomata are rare, myxomata and myxochondromata show a local malignancy, tending to recur after operative removal.

#### 5 Traumatic Cysts

Not unfrequently a radiogram of the carpus taken a few weeks after an injury may show a central cavity in the scaphoid in cases where radiograms taken immediately after the injury have shown no evidence of fracture. These cavities have been loosely termed traumatic cysts. It is not correct to regard these scaphoid cysts as occurring without previous fractures unless extra radiograms have been taken in the oblique plane. It seems probable that many such cysts are the result of non-union of a fine fracture even though no fracture was demonstrated originally.

*Malone* under the title of "Traumatic Cystic Disease of the Carpal Bones," describes five cases of this condition. He regards it as analogous to Kienbock's disease of the semilunar and holds that it is due either to traumatic disturbances in nutrition followed by a progressive cystic change or to an aseptic bone necrosis. The cysts may be found in carpal bones other than the scaphoid.

6 and 7 Infective and Developmental Cysts in Relation to the Teeth are dealt with in another section.

### 8 Arthritic Cysts

In radiograms of joints showing chronic osteoarthritic changes particularly in the hip joints there are seen on occasions small round translucent areas at or near the articular surface of the bone. These may be surrounded by a capsule of denser bone and may appear to communicate with the joint space, occasionally neighbouring translucent areas may be confluent. They are caused by resorption and fibrous replacement of the cartilage and bone and they may contain giant cells.

### 9 Hydatid Cysts

Involvement of bone in hydatid disease may occur either by implantation of the embryo in the bone usually near the epiphyseal ends or by invasion of the bone by an extra-osseous hydatid cyst.

In the former the embryo grows in the line of least resistance along the bony canals. By pressure on small arteries areas of necrosis are produced in which caseous debris may be found. In course of time large areas of cancellous bone may become invaded, degenerated and necrotic. Little or no periosteal reaction is usually observed and perforation of the periosteum takes place, the cyst invading the surrounding soft tissues. Pathological fracture is common with as a rule subsequent non union.

The bones more commonly affected are the humerus, femur, and tibia. Thus both the bones affected and the site of the lesion in them (near the epiphyseal line) correspond to those in which the simple bone cyst may occur. In such cases the lack of bony expansion about the translucent area and the demonstration in it of caseous debris may suggest the presence of a hydatid cyst rather than of a simple bone cyst. Clinically, where a pathological fracture has occurred the absence of severe pain or crepitation or oedema or ecchymosis and the subsequent non union are characteristic of hydatid disease. The presence of an extra osseous soft tissue swelling is confirmatory evidence.

Radiologically the hydatid cyst may simulate a simple bone cyst or even extensive fibrosis (fibrocystic disease) of bone. The absence of any corresponding degree of expansion or of periosteal thickening may give a clue to the diagnosis.

In many cases the diagnosis is made only after puncture of the cyst

Though more common in the long bones it should be mentioned that hydatid disease may affect a flat bone, such as the innominate bone or scapula. In such sites the radiographic appearances may simulate fibrocystic disease, but the presence of a soft, painless, extra-osseous swelling, possibly causing pressure on neighbouring parts such as the rectum, may serve to differentiate them. Supplementary evidence may be obtained from the Casani and complement fixation tests.

In the vertebral column the disease is not uncommon, and may start in any part of the vertebra. Sooner or later it extends beyond the bone, causing various pressure effects according to the site and direction of this extension.

### 10 Fibromatous Cysts (Chronic Fibrous Osteomyelitis)

This is a small localised collection of fibrous tissue in the bone, often surrounded by a capsule of sclerosed bone. Histologically it resembles a bone cyst but *Phemister* regards many of these cases as being the end result of chronic osteomyelitis, thus simulating Brodie's abscess, of which it may represent a late, partially healed, stage.

## BIBLIOGRAPHY

### Tumours and Cysts of Bone

#### *Cysts*

- BLOODGOOD (a) *Ann of Surg*, LII, 163, LXXIII, 712, (b) *Amer Jour Surg*, 1923, XXXVII, 109  
 COLEY and HIGGINBOTHAM, *Ann of Surg*, 1934, 432  
 DEW, Hydatid Disease, 369-389  
 ELMISLIE, *St Bart's Hosp Reports*, 1935, LXVIII, 147  
 GARLAND, *Amer Journ Roentgenol*, 1929, XXII, 517  
 HUNTER, *Quart Journ Med*, April 1931, XC, 393  
 KNAGGS (LAWFORD), "Inflam and Toxic Dis of Bone," 1926  
 PREMISTEP, *Ann of Surg*, 1929, XC, 706

#### *Tumours*

- BENTZON, *Acta Radiol*, III, fasc 2-3, No 12, VII, 13, 1924  
 BRAILSFORD, *Brit Journ Rad*, March, 1937, V, III, 171  
 BUCY and CAPP, *Amer Journ Roentgenol*, January 1930  
 CAMP, *Papers of Mayo Clinic*, 1935, XXVII, 1082  
 CODMAN (a) 'Bone Sarcoma,' Paul Hoeber, New York, 1925, (b) *Surg Gyn and Obstet*, 1926, XLII, 381-393  
 COLEY, (a) *Surg Clin N Amer*, 1929, IX, 593, (b) *Glasgow Med Journ*, 1936, 126  
 ELMER and BOYLAN, *Amer Journ Surg*, XXXIII, 1936, 252  
 EWING (a) *Archives of Surgery*, May, 1922, IV, 485-533, (b) 'Neoplastic Diseases,' 3rd Edition, Saunders, 1928, (c) *Proc N Y Path Soc*, 1924, XXIV, 93  
 FAY, *Surg Gyn and Obstet*, 1914, XIX, 174-190  
 GESCHICKTER and COPELAND, "Tumours of Bone," *Amer Journ of Cancer*, New York, 1931  
 GOLDEN, *Diagnostic Radiology*, Nelson & Sons, 1936

- HANDLEY, *Brit Journ Surg*, 1926, XIII, 762  
 HARRISON (STEWART), *Brit Journ Rad*, 1934, VII, 580  
 HARTMAN, *Surg Gyn and Obstet*, 1922, XXXIV, 161  
 HERTZLEP, "Surgical Pathology of the Disease of Bone," Lippincott Coy, Philadelphia  
 Montreal, and London, 1930  
 HUME, *Brit Journ Surg*, 1929-30, XVII, 236-241  
 JANSEN, "Robert Jones Birthday Volume"  
 KPIILLER, *Surg Gyn and Obstet*, 1925, XL, 510  
 KEFITH, *Journ of Anat*, 1910-20, 101-115  
 KOLODNY, "Bone Sarcoma," Surgical Publishing Co, Chicago, 1927  
 MEYERDING, (a) *Surg Gyn and Obstet*, 1922, XXXIV, 321-332, (b) 'Surg Clin  
 N Amer,' 1921, I, 1500  
 MORISON, *Brit Journ Rad*, April, 1934  
 OGILVIE, *Proc Royal Soc Med* April, 1937, XXX, 6, 803  
 OSCHNER and GAGE, 'Surg Clin N Amer,' 1930, X, 851  
 OSOOD, 'Surg Clin N Amer,' 1921  
 PLATT, (a) Lady Jones Lect, Lpt, *W Ch Journ*, 1935, XLIII, 42, (b) *Proc Roy Soc  
 Med*, 1931, XXV, 1-41  
 REXFORD, *Ann of Surg*, 1930, LCH, 673  
 ROBERTS, (a) *Brit Journ Rad*, March, 1937, X, No III, 196, (b) *Brit Med Journ*, 1937,  
 II, 1207; (c) *Proc Roy Soc Med*, 1926, XIX, 14-39  
 STONE and EWING, *Arch Surg*, 1923, VII, 280  
 WATSON JONES and ROBERTS, *Brit Journ Surg*, 1934, XXI, 83  
 WRIGHT, J Hopkins Hosp Rep, 1900, IX, 359  
 YOUNG, *Brit Med Journ*, 1937, II, 647

## *PART THREE*

### SECTION X

### THE SOFT TISSUES

BY

E DUFF GRAY M A M D F F R D M R E

### CHAPTER XLVIII

#### CALCIFICATION AND OSSIFICATION IN THE SOFT TISSUES

#### GENERAL CONSIDERATIONS

THE RADIOGRAPHIC differentiation of the various structures which make up the normal soft tissues of the body is possible only to a limited extent because there is practically no difference between them as regards the absorption of X rays. The only exception is fat which has slightly less density than the other soft tissues and if present in sufficient quantity can be shown radiographically as an area of increased translucency.

Apart from those regions where soft tissues are demonstrable by reason of the presence of air containing cavities for example in the neck and thorax it is on the presence of fat that we must depend for normal soft tissue differentiation. As a rule fat exists in sufficient amounts to be demonstrable in the subcutaneous tissue and between fasciæ muscles and tendons.

Larger collections of fat are to be found beneath the infrapatellar and Achilles tendons and they are readily demonstrable as areas of increased translucency in lateral views of the knee and ankle.

The ordinary routine film of a limb taken primarily to show bone structure should be of such quality as to show the outline of the skin surface the relative translucency of the subcutaneous fat and the long straight or slightly curved lines of increased translucency which indicate fat in those intermuscular planes which happen to be tangential to the rays. In such routine films the soft tissues are often relatively over exposed and a strong light should be available on the viewing desk for the soft parts to be scrutinised. In this examination one looks for abnormal prominence of the skin surface distortion of intermuscular planes and any evidence of local increased or diminished density. If anything abnormal is seen which requires further explanation greater contrast in the soft parts can be obtained by taking other films with

lower kilovoltage. Several trial exposures are usually necessary to get the maximum differentiation of the required structure.

In soft tissue radiography the optimum kilovoltage is rather more critical than in routine bone exposure. Under penetration is to be avoided. Intensifying screens must almost invariably be used in order to get films in reasonable time. It is advantageous in most cases to do without the upper screen and very often the loss of speed may be compensated by decreasing the tube-film distance. In examining the films variable illumination is frequently helpful.

Low penetration films ought to show, in favourable situations, the fibrous strands running through the subcutaneous fat which serve to anchor the

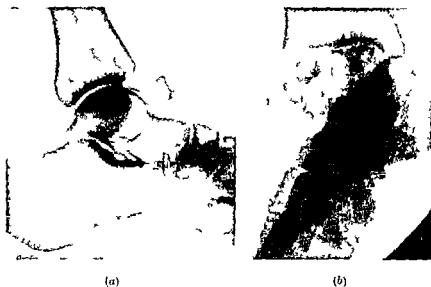


FIG. 529—(a) Wart impregnated with metallic ointment. Tangential view (b) shows the shadow to be on the surface of the skin.

skin to the deeper structures and also several of the larger subcutaneous veins. In somewhat harder films the fascial and intermuscular planes will be made more obvious and any abnormal opacity or translucency will be seen with greater distinctness.

In examining films of the soft tissues special care must be taken to avoid error from artefacts due to such causes as screen and film flaws and processing stains. Clothing, dressings and medicaments on the patient's skin are also liable to be mistaken for lesions in the soft tissues.

Tattoo marks are not common nowadays. Some of the dyes used cause quite appreciable opacities.

Normal skin folds by reason of the air which they enclose, may produce lines of increased translucency which are sometimes puzzling, especially in

films of the hands and feet Superimposed structures such as the pinna and glans cast quite dense shadows which may be misinterpreted

Skin tumours such as warts and sebaceous cysts may cause confusing abnormal opacities Warts with their fissured and granular surface

especially if impregnated with metallic ointments may very closely simulate the trabecular structure of a bony fragment (Fig 529) The shadow of a sebaceous cyst seen in a skull film is often suggestive of a local area of bone sclerosis

Dense opacities in the soft tissues are caused by areas of calcification or ossification Minor degrees of increased opacity are seen in non calcified tumours (excluding lipomata) abscesses (Fig 530) and hæmatomata if of sufficient size The degree of opacity due to foreign bodies depends on their density

Areas of markedly increased translucency are caused by gas or air in the soft tissues A minor degree of increased translucency may be present in lipomata

### CALCIFICATION IN THE SOFT TISSUES

Normally the deposition of calcium in the body is concerned with the formation maintenance and repair of the skeletal system but under certain conditions which are not yet fully understood fibrous tissue and cartilage in any situation may become calcified In the common form of soft tissue calcification which is known as

*dystrophic calcification* the most important factors are undoubtedly local tissue degeneration combined with an impoverished blood supply The sites of this type of calcification are very numerous and include practically every soft tissue structure in the body Inflammatory and tuberculous foci and abscesses hæmatomata parasitic cysts and many forms of neoplasm both simple and malignant are also liable to this type of calcification

Several instances of the occurrence of dystrophic calcification are considered under their appropriate sections as for example calcification in costal and laryngeal cartilages lymph glands pericardium pleura adrenals thyroid adenoma uterine fibroid hypernephroma dermoid cyst hydatid cyst



FIG 530 Tuberculous abscess in belly of biceps Soft tissue film Abscess shown as a rounded shadow in the region between the skin markers



tuberculous foci in various situations and several types of intracranial calcification. In the following pages other examples of dystrophic calcification will be dealt with. In all these instances calcium is deposited in tissues that are of low vitality by reason of previous disease or senescence.

Cases do occur, though rarely, in which calcium is deposited in previously normal tissues. This is known as *metastatic* or *metabolic calcification*. In this form of calcification the calcium is derived from the skeleton, usually as a result of some widespread destructive bone lesion, as for example, in hyperparathyroidism or multiple myelomatosis. The excess of calcium in the blood cannot be excreted sufficiently rapidly and tends to be deposited in such situations as the kidneys, stomach, lungs, arteries, and liver. Cases of this type of calcification have occurred experimentally in hypervitaminosis D produced by toxic doses of irradiated ergosterol.

A third type of soft tissue calcification is known as *calcinosis* (vide p. 654). The aetiology of this condition is still obscure. It is more probably an example of dystrophic calcification, but a metabolic disturbance cannot, as yet, be excluded.

Radiographically deposits of calcium in the soft tissues, if present in sufficient quantity, are readily demonstrable by reason of their marked relative density as compared with the normal neighbouring structures.

The deposits appear as sharply defined dense opacities which may be homogeneous, or may be made up of multiple granular spots or irregular structureless masses. In some instances the calcification may be seen as a plaque or in the form of a ring.

### CALCIFICATION IN ARTERIES

In patients over middle age calcification in the peripheral arteries is extremely common, particularly in the lower extremity. Its radiographic appearances have already been considered in Volume I.

The question of arterial calcification may be the deciding factor in distinguishing between arterio-sclerosis and thrombo-angitis obliterans, the faintest trace of calcification being sufficient to put the case in the arterio-sclerotic group (Telford).

### CALCIFICATION IN VEINS

Calcification in the wall of a vein is excessively rare. It has been recorded in the superficial veins of the leg (Baastrup). This case showed a network arrangement of annular and tubular opacities in the subcutaneous tissues.

Small calcified thrombi, so called phleboliths, are of frequent occurrence in the pelvic veins, though they are relatively uncommon in other situations. They have been observed in the scrotum, labia and spleen, and very occasionally in the veins of the extremities.



FIG. 531. Radiogram and clinical photograph of a case of hæmangioma. Male. Lesion present from birth. Rapid increase in size accompanied by severe pain in last few months. The radiogram shows calcified thrombi in the hæmangioma.

Phleboliths appear radiographically as sharply defined rounded opacities about 2 to 5 mm in diameter. They are as a rule structureless but are sometimes laminated. In the pelvis they are generally arranged in rows rather than in groups.

### HÆMANGIOMA

Calcified bodies resembling phleboliths are seen in subcutaneous cavernous angiomas and in hæmangiomata of muscles. These bodies which vary in

size from 1 mm to 1 cm are rounded or oval and the larger ones frequently show concentric rings. They represent calcification of organised blood clots in dilated vascular spaces (Fig 531).

### CALCIFICATION IN HÆMATOMATA

The normal process of repair in subperiosteal hæmatomata naturally includes calcification and ossification but intramuscular hæmatomata also occasionally calcify and in some cases ossification takes place later as in the condition commonly known as myositis ossificans (*vide infra*).

### CALCAREOUS DEPOSITS IN TENDONS

**Calcareous Tendinitis (so called Subacromial Bursitis)**

The commonest situation for this condition is in the supraspinatus tendon near its insertion into the greater tuberosity of the humerus. The typical radiographic appearance is illustrated in Fig 532 (a) which shows a large dense opacity lying above the outer part of the head of the humerus and extending under the acromion process.

The condition is most frequently met with in patients of middle age



FIG 532—(a) Calcareous deposit in tendon of supraspinatus. (b) Traumatic avulsion of greater tuberosity of humerus for comparison with (a). Tuberosity absent from normal site and lying above head of humerus. The abnormal shadow shows the structure of bone.

There is generally no history of definite trauma but recent over use of the arm may be admitted

Formerly it was supposed that the opacity was due to calcification in the subacromial bursa but recent work has shown that almost invariably it is due to the deposit of calcium in the supraspinatus tendon itself near its insertion

It is quite certain that the clinical symptoms and the radiological findings do not bear a constant relation to one another in this condition For instance there may be symptoms in only one shoulder and yet the condition may be seen radiographically to be bilateral

Spontaneous absorption of the calcareous deposit can occur in a relatively short time—in the case illustrated the opacity was only just discernible on re examination after an interval of two months Sometimes the calcareous material is dispersed into the overlying bursa and it may be rapidly absorbed from there

The differential diagnosis from avulsion of part of the greater tuberosity of the humerus is as a rule not difficult The opacity in such a case is clearly of bony structure and the defect in the greater tuberosity can be recognised (Fig 532 (b))

Other tendons in the region of the shoulder which may show similar calcareous deposits though much less common are the infraspinatus, teres minor and very rarely the subscapularis

In the region of the greater trochanter of the femur the tendon of the gluteus medius may be affected and more rarely that of the piriformis (*Goldenburg and Leventhal*)

### SOFT-TISSUE CALCIFICATIONS DUE TO PARASITES

Several parasites occurring in the soft tissues in man are known to undergo post mortem calcareous degeneration Whether they may be demonstrated radiographically depends on their size and on the degree of calcification which has taken place

#### *Cysticercus Cellulosæ*

In this country the only calcified parasite likely to be found is the *cysticercus cellulosæ* and even this is extremely uncommon and is practically confined to soldiers who have had service in India The condition is of importance on account of the cerebral manifestations of cysticercosis The subject has of recent years been clarified by the work of *MacArthur, Dixon* and *Smithers* and others

In Asia and in certain parts of Europe the adult stage of *tænia solium* occurs as a not uncommon tapeworm in the intestinal tract of man The larval stage is usually passed in the pig which is infected by eating garbage contaminated by tapeworm ova from human dejecta The ova develop

into embryos in the intestine of the pig, and migrate to its muscular system and other tissues, where they become encysted. The normal cycle is completed when man eats measly pork and a tapeworm is once more developed.

Man may, however, act as the intermediary host if ova enter the stomach. This may occur by "man-to-man" infection, or possibly through self-infection by ingestion of the ova of the patient's own tapeworm; man does not contract cysticercosis from eating measly pork. After ingestion of the ova the process develops as in the pig. The embryos penetrate the intestinal mucous membrane and are carried by the blood to different parts of the body, where they settle in the tissues. The most common sites are the voluntary muscles and the brain. Within a few months the cysticerci become encapsulated, but they may remain alive for several years, probably three or four at least, and during this period give little or no indication of their presence. When they die, however, there is local tissue reaction from the evolution of toxic products of degeneration, and also pressure effects from the post-mortem swelling of the cysts.

In the brain, these changes at the time of death of the cysticerci bring about epileptic fits of Jacksonian type, and less commonly other cerebral disturbances, while in the tissues small subcutaneous and intramuscular nodules may in some cases become palpable. These nodules disappear within a short time. At this stage the cysts are not demonstrable radiographically.

The cysticerci, having died, tend to calcify. The process is a slow one, and it is estimated that it takes about three years for the occurrence of sufficient calcification to be visible radiographically. It is to be noted that calcareous degeneration rarely takes place in the cerebral cysts—in rather less than 10 per cent. of cases, according to *Smithers*.

The patient, as a rule, comes under medical care for cerebral symptoms, and if cysticercosis is considered as a possible cause, the X-ray examination should not be confined to the skull (for the findings here will generally be negative), but must include a careful search for any abnormal calcifications in the soft tissues of the whole body.

Cysticerci, when calcified, are seen radiographically as dense, somewhat elliptical bodies, lying in the muscles with their long axes in the direction of the fibres. *Morrison* distinguishes twenty-five different shapes that may be seen. By far the most common is the elliptical, with one end rather more pointed.

The shadows vary considerably in size, from mere dots up to 25 mm. in length, the average being about 15 mm. long and 3 mm. wide. As a rule, they are not homogeneous. Some have an irregular crust of calcification, and practically all have a central non-calcified area which may be seen as a circle in those cysts which happen to lie end-on.

The above description applies to the intramuscular shadows. In the unusual cases where calcification occurs in the cerebral cysts, they are seen



as small rounded opacities a few millimetres in diameter and quite few in number. *Dixon and Stillers* describe such a case.

Fig. 533 shows the typical opacities in the case of a male aged 36 who had for six years suffered from fits of Jacksonian type. The calcified cysticerci were widely distributed throughout practically the whole of the voluntary muscular system but were most numerous in the legs and thighs. There were none in the hands and feet and few in the forearms.

An x-ray examination of the skull was entirely negative but it was demonstrated at operation that several non calcified cortical cysts were present.

An absence of intramuscular calcifications does not negative the diagnosis of cysticercal epilepsy because sufficient time may not have elapsed for the calcification to have taken place. *MacArthur* points out that a positive radiological finding is not to be expected if the patient's nervous symptoms are of less than four or five years duration. An x-ray examination may therefore have to be repeated at intervals over a period of years before positive radiological evidence is obtained. In rare cases cysticerci are present exclusively in the brain and as these rarely calcify there may be no possibility of radiological confirmation.

#### *Trichina (Trichinella) Spiralis*

In man trichinosis (or trichiniasis) is generally acquired by the ingestion of infected pork.

The condition is by no means un-

FIG. 533.—Calcified cysticerci in muscles of leg

common in the United States. *Hall* found an incidence of 12.5 per cent. in reviewing 1,778 post-mortem records. In this country, the frequency is probably much less. *Van Someren*, working in London, found evidence of infestation in two cases out of 200 specimens examined.

The clinical effects are extremely varied, and the condition is rarely diagnosed during life, owing to the limited value of the diagnostic tests at present available.

The larvæ become encysted in the muscular system, most commonly in the diaphragm and intercostal muscles, and after their death calcification takes place in the cysts. The time required for calcification has not yet been established. It is probably a period of years. Irradiated ergosterol is known to hasten the process. *Wanland* has shown that if artificially infected rabbits are fed on irradiated ergosterol and calcium lactate, a degree of calcification normally found after eight to twelve months is obtained within three months.

The size of the calcified cysts is not more than .5 mm., and it is therefore very improbable that they can be demonstrated radiographically during life.

*Brailsford* has suggested that an attempt should be made to demonstrate part of the masseter muscle on a dental film as affording the best chance of showing the cysts. The writer has recently radiographed a museum specimen of trichinosis after placing the piece of muscle in close contact with a fine-grain film. The resulting radiogram, when examined by transmitted light, showed innumerable calcified cysts, but they were of such small size as to be only just discernible without a magnifying-glass.

The author of a recent article on trichinosis made the statement that in one case several small calcified trichina cysts showed well in a chest radiogram, but this cannot be accepted, since subsequent examination of the records of this particular case left no doubt that the visible calcifications were cysticercus cysts.

In a personal communication *Dr. B. R. Kirklin* stated that there was no record of a case having been recognised radiographically at the Mayo Clinic.

### **Filaria Bancrofti**

This worm causes lymphatic obstruction, the two chief manifestations being hæmatochyluria and elephantiasis.

Adult filariæ, when they have undergone post-mortem calcification, may be demonstrated radiographically. *O'Connor*, *Golden*, and *Auchincloss* state that the shadows vary in size, from small dots to elongated forms 5 mm. by 1 mm. The opacities are situated in the subcutaneous fat of the leg or thigh and are by no means easy to demonstrate. Typically, they form chains of small opacities, but they may occur singly or in irregular groups. The authors quoted suggested that the worm shadows should be looked for in the tender areas, because it has been shown that calcified and live worms are often found in the same region.

**Guinea Worm (*syn* *Dracunculus Medinensis*, *Filaria Medinensis*)**

The female worm is generally found in the subcutaneous tissue of the foot or leg. It may attain a length of 60 cm or more. As a rule, the worm is



FIG. 534.—Calcified Guinea worm in popliteal space

spontaneously extruded through the skin, but if retained, it may undergo calcification, as in the case illustrated in Fig. 534. This shows in the popliteal region a single worm, which has calcified in some sections of its length. The patient had lived in a district of India where the disease is common though he had not suffered any disability from the infection.

Connor points out that irritative local lesions due to calcified pieces of the

dead worm remaining buried in the tissues are not uncommon, and that surgical excision may be necessary.

**CUTANEOUS AND SUBCUTANEOUS CALCIFICATION**

Cutaneous and subcutaneous calcification is relatively uncommon. Certain skin tumours may occasionally calcify, such as fibroma, epithelioma and sarcoma and so also may old lupus nodules. Sometimes a few small subcutaneous calcified bodies may be found on the inner aspect of the tibia in old people—these are considered to be calcified fat lobules. Tumours of supposed sebaceous gland origin may calcify. These occur chiefly in women and children (*Sequeira*). Skin calcification may also be found in cases of metastatic calcification.

**Calcinosis**

This rare condition occurs in two forms—the localised and the diffuse.

**Calcinosis Circumscripta** (*Kalkgicht*, *Hypodermolithiasis*) usually appears in middle age and is more common in females than in males. It is characterised by the presence of localised granular calcareous deposits in and under the skin. The most common site is in terminal phalanges of the fingers on the flexor aspect. Many of the recorded cases have had associated vasomotor disturbances of the hands and about one third have shown scleroderma.

Radiographically, the opacities are nodular and extremely dense. The



bones and joints in the neighbourhood appear normal. Other regions of the body should be examined to determine the extent of the disease.

An unusual site is in the skin of the scrotum (Fig. 535) (*Tate and Trumper*).

*Calcinosis Universalis* is usually found in children and young adults. It is a serious condition and often fatal. Widespread calcareous deposits are present as large plaques in the skin and subcutaneous tissue and there may also be calcification in the interstitial connective tissue of deeper structures—tendons, muscles, and nerve sheaths. Calcified masses may occur in the neighbourhood of the larger joints.

There are superficial resemblances between this condition and *myositis ossificans progressiva* in that both are found in young patients, both are gradually progressive, and both show widespread abnormal opacities radiographically. In *myositis ossificans* however, there is bone formation, chiefly in the trunk muscles, and in most cases there are associated developmental anomalies. In *calcinosis* there is always extensive skin calcification even if the deeper structures are involved. The distinction between the two conditions should not give rise to any real difficulty.



FIG. 535.—*Calcinosis circumscripta* affecting skin of scrotum.



FIG. 536. Ossification in long plantar ligament (Note also calcaneal spur).

## OSSIFICATION IN THE SOFT TISSUES

The presence of bone in the soft tissues is readily explained when there has been rupture of the periosteum, with displacement of bone-forming cells. It is, however, possible for bone to be formed in tissues which have no connection with the osseous system. This heterotopic ossification is a rare phenomenon, and has been the subject of much discussion.



FIG 537 Heterotopic ossification. Tangential view of bone in laparotomy scar taken eight months after the operation.

The process was inexplicable so long as it was supposed that bone could be produced only by a specialised cell—the osteoblast. It is now believed that in certain instances the ordinary connective tissue fibroblast is capable of bone production.

Heterotopic ossification occurs only in fibrous connective tissues in which calcium has been previously deposited (Fig 536). Thus it may occur in any of the sites of dystrophic calcification already mentioned though it is by no means common.

An interesting example of heterotopic ossification is in the scar of an abdominal incision (Fig 537). The bone is formed as a thin plaque and generally requires tangential views to be demonstrated radiographically.

Bone formation in the connective tissue of muscles and tendons is considered later.

The factors which bring about heterotopic ossification are not fully understood. *Watson Jones* and *Roberts* in a recent review of the subject lay great stress on the blood-supply of the region concerned. They state that whereas pathological calcification may occur in any mesenchymatous tissue of low metabolism when the blood supply is decreased by reason of the fibrosis of trauma or infection subsequent ossification demands an adequate blood supply. The

stimulus to revascularisation may be trauma or possibly irritation caused by granules of calcium.

Radiographically the change from calcification to ossification may be extremely difficult to recognise. It is only possible when definite bony trabeculation can be demonstrated. The radiographic density of bone is in general less than that of a deposit of amorphous calcium of equal size.

### OSSIFICATION IN TENDONS

Bony spurs are often present at tendinous and ligamentous insertions. The common sites are the os calcis, olecranon, patella and external occipital protuberance. These spurs are considered to be due to local elevation of the periosteum by traction with subsequent bone production by cells derived from the periosteum itself. They are not therefore to be regarded as examples

of heterotopic ossification. The process of spur formation is a gradual one and is rarely of clinical importance.

Ossification has been found occasionally in a tendon remote from its insertion. Watson, Jones and Roberts give references to eight cases



FIG. 338.—Ossification in flexor tendon of index finger the result of occupational trauma.



FIG. 371.—Ossification in muscles around hip joint occurring in a male of 45 paralysed for two years.

of heterotopic ossification in the tendo Achillis and record two further cases in the same tendon. Subcutaneous tenotomy had been carried out in most of the recorded cases many years previously and the presumption is that fibrosis was followed by calcification and subsequently by the formation of bone as a result of revascularisation.

Repeated minor traumata may also lead to ossification in a tendon and tendon sheath as illustrated in Fig. 338. In the course of her work this patient had to press heavily with the index finger on the handle of a knife over a period of many years.

### SOFT-TISSUE OSSIFICATION IN SPINAL CORD LESIONS

In some cases of complete transverse lesions of the spinal cord there may be extensive bone formation in the tendons, muscles and joint capsules of the leg leading to ankylosis as in myositis ossificans progressiva (*Schinz, Baensch and Friedl and Lasker*). This type of ossification is illustrated in Fig 53.)



FIG 54. Chronic myositis ossificans brachialis anticus following a proximal fracture of humerus.

### TRAUMATIC MYOSITIS OSSIFICANS (TRAUMATIC OSTEOMA)

In this condition the resolution of an intramuscular hæmatoma proceeds abnormally and bone is formed between the muscular fibres. In

some cases this bone formation may be due to osteoblasts displaced as a result of periosteal rupture but such a mechanism need not be postulated in all cases since it is known that the ordinary connective tissue fibroblast is capable of bone production although the factors which bring about the metaplasia of fibrous tissue to osseous tissue are as yet unexplained. The two muscles most commonly involved are the brachialis anticus and the quadriceps femoris. In the brachialis anticus the lesion generally follows a posterior dislocation of the elbow or a fracture in this neighbourhood (Fig 54) and in the quadriceps it is generally the result of a severe contusion such as a kick at football.

Radiologically there is no demonstrable abnormality in the region of the muscle until at least two to three weeks after the injury when a homogeneous opacity with ill defined

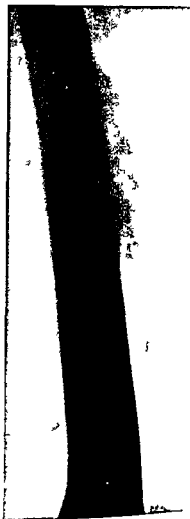


FIG 55.—Traumatic myositis ossificans of quadriceps. Male 16. Severe kick on thigh ten weeks previously. Hard palpable mass attached to femur at its upper end.

margin is seen, generally in close relation to the underlying bone, but separated from it by a definite space. Films taken at intervals of a few weeks will show the gradual transformation of the opacity to a rather dense, clearly defined plaque of bone, irregular in structure and showing linear streakiness or lamination (Fig 541). In some cases there may be numerous spots of increased density in parts of the mass. The new bone is generally of considerable size, especially in the thigh, and lies roughly parallel to the neighbouring shaft remaining entirely or to a large extent distinct from it. The cortex of the underlying shaft shows no abnormality, but occasionally slight periosteal reaction may be seen. The amount of bone formation may increase for a time but after some months the mass tends to diminish in size, while increasing in density and sharpness (Fig 542). After an interval of about six to nine months it generally remains stationary though complete spontaneous disappearance some times takes place.

Surgical removal of the new bone may be necessary. In this case it is important to make sure by serial radiograms that the bony mass has reached a stage of quiescence, otherwise recurrence is almost certain.

Repeated minor traumata may also result in local intramuscular ossification, as in the adductor muscles in the condition known as "rider's bone."

### MYOSITIS OSSIFICANS PROGRESSIVA

This is a rare disease of unknown aetiology which appears early in life, generally before the sixth year, and is characterised by a progressive ossification of the connective tissue in skeletal muscles. The ossification appears, as a rule, in the muscles of the neck and upper back, and spreads to the arms, trunk, and legs. Most of the bony masses are attached at some point to the skeleton, and consist of elongated

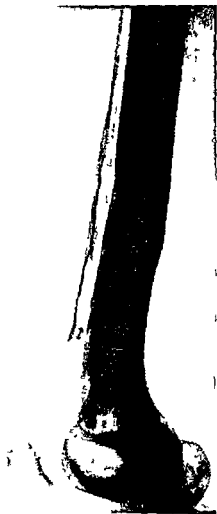


FIG 542—Myositis ossificans. Male, 15. Three months after osteotomy of femur which healed well patient required manipulation to restore full movement of knee. During ensuing nine months a 'flocculent' mass of bone formed in the quadriceps this gradually became reduced to half its original width denser, and more compact, as shown in above radiogram—quiescent stage.

plaques lying in the long axis of the affected muscles. Ossification also involves perimuscular connective tissue, tendons and fasciæ.

In a large percentage of the reported cases there has been a developmental anomaly of the great toes and thumbs. Microdactylia is present with fusion of the phalanges and the great toe shows valgus deformity.

In the case reported by *Mather* the condition was first demonstrated radiographically at the age of 2½. The patient became bed ridden when she was 15 years old and by the time she was 36 years of age there was very widespread ossification in the voluntary muscular system (Fig. 543).



FIG. 543. Myotonic fevers: progressive in a woman aged 36. Condition present from infancy (see text above).

### PELLEGRINI STIEDA LESION

In this condition a curved opacity of bony structure is seen lying in close relation to the upper part of the internal femoral condyle (Fig. 544). In most cases there is a history of trauma at least three to four weeks previously. So far there is not complete agreement as to the causation of this shadow. It is certainly not due to the detachment of a fragment of bone since it is never seen immediately after an injury and there is no corresponding defect in the outline of the condyle. The general view is that the opacity represents a local

post traumatic ossification in connective tissue but whether the bone is derived from injured periosteum or whether it is due to metaplasia has not been settled.

In a recent case fully reported with operative findings by *Collart* the lower end of the bony flake was firmly connected to the superficial layer of the internal lateral ligament of the knee joint and the upper end passed above the internal condyle in front of and separate from the adductor magnus tendon. The new bone was completely covered with fibrous tissue which was adherent to the internal condyle.

Serial examinations of individual cases have shown that the shadow may vary in size and density and that it may even disappear. In *Collart's* case mentioned above the shadow recurred two months after operative removal of the bony fragment. *Brailsford* illustrates a case which had in addition a similar though much smaller shadow parallel to the inner tuberosity of the tibia.



FIG. 344.—Pott's lesion on

## CHAPTER XLIX

### OTHER LESIONS OF THE SOFT TISSUES

#### GAS IN SOFT TISSUES

GAS in the soft tissues produces localised areas of markedly increased translucency and is readily demonstrable radiographically even when present in quite small amounts. The local translucency due to collection of fat in normal situations and in deep lipomata is not nearly so great as that due to gas and should not cause any difficulty in differential diagnosis. One must of course always exclude artefacts due to dressings, flaws in screens and films etc.

The most important cause of gas in the soft tissues is gas gangrene. Other possible causes are escape of air from the lung or trachea as a result of penetrating wounds and lacerations; penetrating wounds of the nasal cavity and accessory nasal sinuses; perforation of the pharynx or œsophagus by a foreign body; gas inclusions in compound fractures and lacerated wound; irrigation of wounds or sinuses with hydrogen peroxide. Exploratory puncture of the soft tissues for abscess might conceivably leave some gas in the tissue. A very rare cause is gas derived from the alimentary tract. *Vorrich* has reported such a case in which an iliopsoas abscess resulted from perforation of the appendix. Radiographically this showed a large collection of gas in the region of the femoral neck and lesser trochanter. Air may be artificially introduced into the tissues for diagnostic purposes as for instance into the breast (see page 666) or into a synovial cavity for the radiological exploration of a joint.

#### Gas Gangrene

Although uncommon in civilian practice this condition must be borne in mind when examining radiograms of compound fractures and injuries associated with penetrating foreign bodies and extensive laceration. If on examining a radiogram in such a case there is any suspicion of gas in the soft tissues the matter should be regarded as urgent and the clinician must be informed at once.

Gas gangrene occurs in wounds infected by a group of anaerobic bacilli which in suitable conditions have the property of producing rapid putrefactive changes in muscle with the evolution of gas. The infection is usually a mixed one, the most common organisms being *B. welchii* and *B. sporogenes*.



Appreciable quantities of gas may be present within a few hours of the injury or the infection may remain latent for several days.

Radiographically, there are two main types of appearances.

(1) **LOCALISED**—The gas may appear as an isolated area of translucency, in strong contrast to the surrounding soft tissue shadow. More commonly a number of discrete bubbles are present, which are mostly oval in shape and may be of various sizes. Sometimes the bubbles are arranged one above the other in a string extending longitudinally in the direction of the muscle fibres. The collections of gas may be at some distance from the track of the original wound. Serial films at intervals of a few hours would show increase in the number of bubbles extending over a large area.

(2) **DIFFUSE**—In this type there is extensive and massive infiltration of gas in the soft tissues. As a rule, the subcutaneous tissue is separated from the underlying muscles by a wide band of translucency and in addition to bubbles of various sizes there is a coarse streakiness chiefly longitudinal in the muscles with rather wider bands of translucency in the intermuscular planes (Fig 545).



FIG 545.—Gas gangrene foil wing (C. S. W. (F.B. in situ).

Savill has described cases

in which there were areas of gas infiltration in the form of fine striations mapping out the individual muscle fibres. The prognosis in such cases was considered to be particularly unfavourable. Well illustrated articles on gas gangrene, based on experience in the war, have been published by *Morison*, *Savill Berry*, and *Morgan and Villandre*.

In the well established case with widely distributed collections of gas, the appearances are unmistakable, and, of course, the clinical signs are equally definite. The help which X ray examination affords in such cases is to indicate the extent of the process, which may be shown to be greater than the clinical indications would suggest, and the X ray findings may influence the type of surgical treatment that must be adopted.

The chief difficulty in interpretation experienced by war time radiologists was the translucency due to traumatic loss of muscle substance—a correlation of the clinical findings would naturally avoid any mistake from this appearance.

In the early case in which relatively little gas is present certain other possible fallacies have to be considered.

Gas bubbles due to inclusion of atmospheric air in wounds are generally

not so clearly defined because unlike gas evolved by organisms they are not under pressure. These air inclusions are found in the track of the wound and disappear within ten to twelve hours. It may be impossible to differentiate gas collections due to  $H_2O_2$  if this has been used as an irrigation. It is to be noted that these collections may be seen at some little distance from the track of the wound.

Experience in the war proved that in some cases the X-ray evidence of the presence of gas infection was obtained earlier than the clinical evidence and the radiologist should therefore not hesitate to inform the clinician at once if there is any radiological suspicion. The results of delay even of a few hours are so serious that it is better to be too active in this regard.

### Subcutaneous Emphysema (Surgical Emphysema)

Following crushing injuries of the thorax in which laceration of the pleura and lung has occurred large quantities of air may escape into the subcutaneous tissues chiefly in the



FIG. 646.—Accidental surgical emphysema following laceration of artificial pneumothorax.

chest wall and neck but possibly extending over the whole body. Radiographically there is massive infiltration of air in the form of streaks and bubbles chiefly in the subcutaneous regions but also in the intermuscular planes. There are no intramuscular collections and no delineation of individual muscular fibres. While the X-ray appearances of the tissues are similar to those of gas infection the clinical findings in the two conditions are entirely different. Subcutaneous emphysema *per se* is a condition of little

clinical importance and clears up spontaneously within a few days. Most commonly the laceration of the lung is caused by fractured ribs and it is interesting to note that a coincident pneumothorax is not necessarily brought about.

Subcutaneous emphysema may occur accidentally during the induction of artificial pneumothorax or as a result of exploratory puncture of the chest especially when the lung has been penetrated in a part which has become adherent to the chest wall (Fig. 346).

Wounds involving the upper respiratory tract and the accessory nasal sinuses may also produce subcutaneous emphysema and it is possible for the condition to occur following a mediastinal emphysema—this has already been discussed in Volume I.

### FAT IN THE SOFT TISSUES

As already mentioned fat is slightly more translucent to X rays than the other soft tissues and its presence allows of the delineation of muscle and tendons. It also shows up by contrast the more opaque subcutaneous fibrous strand. The excessive intermuscular fat in pseudohypertrophic myopathy may be shown radiographically. A lipoma may be sometimes successfully demonstrated as a well-defined rounded or oval area of increased translucency lying between the muscular shadows (Fig. 347). This appearance is characteristic of a lipoma and will differentiate this tumour from other soft tissue neoplasms. The degree of relative translucency depends on the size of the lipoma and also on the proportion of fatty tissue present. If there is much fibrous tissue in the stroma the density of the shadow may be indistinguishable from that of the neighbouring soft parts.

### BURSITIS

Inflammatory changes in a bursa may sometimes be recognised radiographically when the distended sac is seen in profile by the presence of an oval homogeneous opacity in the known position of a bursa. Enlargement of a subcutaneous bursa such as the prepatellar or olecranon will also produce very evident localised projection of the soft tissue outline of the part. In

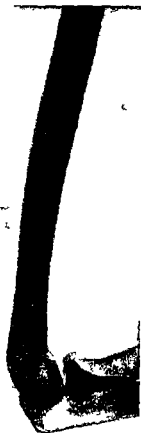


FIG. 347.—Interosseous lipoma of arm shown as a relatively translucent area of thin translucency between the shadows of the muscles.

chronic cases there is frequently roughening of the bone surface deep to the bursa as a result of periosteal reaction

In old standing cases of chronic bursitis there may be calcareous deposits in the wall of the bursa or in loose bodies within it. This is however a relatively uncommon cause of soft tissue calcifications in the neighbourhood of the joints. The well known condition in which calcification appears in the soft tissues above the head of the humerus is now considered to be due almost invariably to calcareous deposits in the supraspinatus tendon and not to calcification in the subacromial bursa as was previously supposed

### RADIOGRAPHY OF THE BREAST

A ray examination of the mammary gland is rarely carried out in this country as it is believed that little information of clinical value can be obtained by this method

In the American radiological literature however there are frequent references to radiography of the breast and a very complete survey of the subject including the most recent technical advances is to be found in an article by *Hicken* and others based on a study of over 600 cases

The most usual method of demonstrating the breast radiographically is to use the supine oblique position with the breast to be examined supported on an angle board having an inclination of about 20 degrees. The central ray is directed tangentially to the chest wall in order to avoid overlapping rib shadows and also because it is important to show the retromammary clear space. The penetration required is usually between 40 and 60 K.V. and a fine focus tube is advisable. Stereoscopic films using a longitudinal shift are generally taken. A more elaborate method in which the breast is made to hang downwards through a slit in a canvas cradle supporting the chest has been described by *Geishon Cohen*.

A radiogram of the normal breast will show a subcutaneous layer of relative translucency due to fat and a similar layer of translucency separating the breast stroma from the chest wall. Between these relatively clear areas is visible the triangular shadow of the breast substance which appears as a moderately dense opacity traversed by denser streaks converging towards the nipple the latter representing normal fibrous tissue strands and the lactiferous ducts. If much fat is present within the breast stroma areas of increased translucency will be present giving a rather mottled appearance. Variations of the normal pattern have been described in the different stages of the menstrual cycle and during lactation.

Malignant tumours cast denser shadows than breast tissue and cause obliteration of the normal striations in the affected area. They are irregular in outline, and often show a featherly appearance on the periphery. Extension of the infiltrating growth may be indicated by local projection of the opacity in the form of thick strands.

Non malignant tumours with the exception of lipomata are also denser than the breast tissue. They have a sharply defined outline and it may be possible to demonstrate that the normal striations are distorted by pressure and not obliterated as in the case of malignant neoplasms. Lipomata produce localised areas of increased translucency.

Hicklen and his co-workers (*loc cit*) show that by using thorotrast as a contrast medium to inject several of the larger lactiferous ducts and by the injection of gas ( $\text{CO}_2$ ) into the retromammary and subcutaneous regions a very complete visualisation of the anatomy of the breast can be obtained. These methods which they describe as contrast mammography have been valuable both in studying the normal breast and in differentiating neoplasms. The procedures are stated to be well tolerated by patients.

### OPACITIES IN THE SOFT TISSUES DUE TO THERAPEUTIC INJECTIONS

In the gluteal regions and thighs and rarely in other situations opacities of metallic density may be observed which are the result of previous intramuscular injections of radio opaque substances such as mercury, bismuth, lipiodol etc.

Mercury remains in the tissues for many years. Cases have been observed in which dense shadows were still present thirty years after the last injection.

Intramuscular bismuth injections tend to disappear relatively quickly—usually within a few months.

Cases have been recorded in which calcification has taken place at the site of previous injections. It is assumed that local necrosis has occurred and later calcium has been deposited in the resulting scar tissue. *Leeser* describes this calcification after the injection of arsenical and bismuth preparations for congenital syphilis in infants and *Linden* records two cases following quinine injections for malaria.

Opacities due to localised deposition of calcium in the soft tissues may occasionally be seen radiographically a few weeks after the intramuscular administration of calcium gluconate—a substance which is not radio opaque at the time of injection.

*Dr C. P. Lapage* has described to the writer two cases of such calcification occurring in infants who had been given intramuscular calcium gluconate for tetany. In these cases hard swellings became palpable at the sites of injection within three weeks and radiograms showed corresponding calcareous masses. Gradual absorption took place during the ensuing three months.

### FISTULÆ AND SINUSES

It is frequently possible to obtain information of great clinical value by outlining fistulous tracks and sinuses with a radio-opaque medium. This procedure is most commonly required to determine the point of origin and

course of a discharging sinus in cases of chronic osteomyelitis and tuberculous disease of bone

Other applications of the method arise in connection with the salivary glands, respiratory and alimentary systems, and the urinary tract. These are dealt with under their appropriate sections. Certain fistulæ of developmental origin are considered later in this chapter.

In outlining a sinus, the use of an opaque tube, such as a ureteric catheter, is rarely successful as it is generally impossible to overcome the tortuosity of the sinus and, in any case, no information is given of ramifications or communicating abscess cavities.

A fluid or semi-fluid medium is almost invariably necessary. Lipiodol is one of the most useful media, especially its colloidal emulsion. The chief difficulty is to inject under sufficient pressure to demonstrate the whole track, while at the same time avoiding reflux of the fluid through the skin opening. As a rule this difficulty can be got over by having available short lengths of rubber tubing of various calibres and using a suitable size to fill effectively the first few inches of the sinus. The tubing may then be connected to a record syringe and the injection made while applying pressure in the region of the superficial opening. When reflux of the fluid finally takes place, the rubber tube is withdrawn and the orifice sealed with collodion or adhesive plaster, taking care to leave as little opaque medium as possible on the skin surface. The site of the opening may with advantage be marked with a circle of wire to facilitate orientation of the track when the radiograms are being examined.

The radiograms necessary depend on circumstances. Two views at right angles are generally sufficient, but oblique views and stereoscopic pairs may be of assistance.

*Brailsford* suggests the use of a semi solid mixture of vaseline containing 25 per cent barium or bismuth. The mixture is heated and allowed to cool until it forms a rather thick paste for injection.

*Schinz* recommends the use of thin pencils of cacao butter containing bismuth. These are made up in lengths of 8 cm., and can be inserted with forceps into the sinus one after another as melting takes place at body temperature. The solid part of the stick serves to block the skin opening.

Certain fistulæ of developmental origin may usefully be examined radiologically.

### Thyroglossal Fistula

The fistula occurs when a thyroglossal cyst breaks through the skin. The external opening is variously situated, as a cyst may be present at any point along the course of the thyroglossal duct. The duct, which is concerned with the development of the thyroid gland, passes exactly in the middle line from the foramen cæcum of the tongue to the upper border of the thyroid

cartilage, where it deviates slightly to one or other side. The duct passes in front of the hyoid, but has a short recurrent loop in relation to the posterior aspect of this bone. *Ruckenstein* has recorded two cases in which X-ray examination was carried out and served to demonstrate the extensive ramifications of the fistula.

### Branchial Fistula (Cervical Sinus)

This fistula, which may be bilateral, is due to faulty development of the branchial arches. The external opening is usually close to the anterior border of the sterno-mastoid, just above the sterno-clavicular joint, and the track extends up to the region of the tonsillar fossa. It does not communicate with the pharynx.

### Preauricular Fistula

This rare fistula is considered to result from aberrant coalescence of the tubercles from which the pinna is developed. The external opening is situated in the crus helix. In a group of cases recorded by *Stammers* the track was almost straight, and extended downwards and forwards for only a short distance. X-ray investigation in such cases would not have been of value, but more complicated tracks have been recorded, as in a case described by *Fournier*, in which a preauricular fistula traversed the parotid gland and reached to below the angle of the jaw. In this particular case X-ray examination would have been useful, for several operations were carried out which might not have been necessary if the full extent of the fistula and its ramifications had been determined in the first instance.

### Coccygeal Sinus (Pilonidal Sinus)

In this condition, which is of developmental origin, one or several minute orifices are present in the middle line posteriorly about the level of the first piece of the coccyx. These orifices are lined by true epithelium and communicate with a superficial cavity which also has an epithelial lining.

In early adult life there is a tendency for the fistula to become infected, with the result that a sinus track is formed which extends upwards and outwards to open on the skin surface in the mid sacral region one inch or more from the middle line.

*Neuell* has recorded eleven cases of this condition in which preoperative X-ray investigation after lipiodol injection of the track proved of value in determining the course and extent of the sinus.

### Umbilical Urinary Fistulae

Urinary fistulae of developmental origin may occur with an external opening at the umbilicus. Several types have been described, and in the more

obscure cases X ray examination has been of great value in determining the exact variety of developmental abnormality. For this examination the bladder is filled *per urethram* with opaque fluid and the fistula is similarly outlined by injection through the external opening.

According to *Begg*, who discusses the developmental aspect in detail information must be obtained on the following points "Does the apex of the bladder itself reach to the umbilicus? And if so, is the upper segment narrowed in the form of a canal representing a partially formed urachus?"

*Begg* states that in cases where the bladder is normally placed, the urine escapes from it either through the dilated terminal portion of the urachal canal or through the weak point at the junction of the urachus with the bladder. A fistulous track extends from the point of escape to the umbilicus passing between the peritoneum and the transversalis fascia.

#### REFERENCES

- BAASTRUP C I *Acta Radiol*, 1932, XIII, 206  
 BEGG, R C *Surg Gynec Obstet*, 1927, XLV, 165  
 BERRY H M, *Proc R Soc Med*, 1916 17, X, Pt II, 17  
 BRAILSFORD J F X ray Diag of Animal Parasites in Man, *Proc R Soc Med Electrother Sec* 1925-6, 19 41 *Radiology of Bones and Joints*, London 1935  
 COLTART W, *Proc R Soc Med*, 1938 XXXI, 3, 180  
 CONNOR E P, *Brit J Surg*, 1922-3 X, 253  
 DIXON, H B F and SMITHERS D W, *Quart J Med*, 1934, XXVII, 603  
 FOURNIER G, *Rev de Laryng*, 1919, XL, 145  
 GERSHON COHEN, J *Radiology*, 1937, XXVIII 2, 234  
 GOLDENBURG R R, and LEVENTHAL S, *J Bone Jt Surg*, 1936, XXIII, 205  
 HALL M C, *Publ Hlth Rep*, Wash, 1937, LII, 16, 468, *Ibid* 1937, LII 17, 512  
*Ibid*, 1937, LII, 18 539  
 HICKEN, N F, BEST, R R HUNT, H B, and HARRIS, T T, *Amer J Roentgenol*, 1938, XXXIX, 321  
 LASKER, W, *Fortschr Geb Rontgenstr*, 1928, XXXVII, 6 830  
 LEFSER, F, *Fortschr Geb Rontgenstr*, 1928, XXXVII, 4, 486  
 LINDEN, O, *Acta Radiol* 1932, XIII 57  
 MACARTHUR W P *Trans R Soc Trop Med Hyg*, 1933 4, XXVII, 4 *Brit Med J* 1935 II 1229 *Brit Encyclopaedia of Medical Practice*, London 1937  
 MANTHER J *Br J Radiol*, 1931, IV, 207  
 MORGAN J D and VILVANDRE, G *Brit Med J*, 1917, I 8  
 MORISON, J M W *Arch Radiol Electrother*, 1915 16 20, 222  
 MORRISON, W K, *Brit Med J*, 1934, I, 13  
 NEWELL, R L, *Brit J Surg*, 1933, XXI, 219  
 NORRISH, R E *Brit J Surg* 1936, XXIV, 55  
 O'CONNOR F W GOLDEN, R, and AUCHINCLOSS, H, *Amer J Roentgenol* 1930, XXIII 494  
 RUCKENSTEINER, L *Fortschr Geb Rontgenstr*, 1936 LIV, 321  
 SAVILL, A, *Proc R Soc Med*, 1916-17, X, Pt II, 4  
 SCHINZ, H R, BAENSCH, W, and FRIEDL, E, *Lehrbuch der Rontgendiagnostik* Leipzig 1932  
 SEQUEIRA, J, 'Diseases of the Skin, London, 1927



- SMITHERS, D. W., *Lancet*, 1937, I, 1016  
STAMMERS, F. A. R., *Brit. J. Surg.*, 1926, XIV, 359.  
TATE, D. C., and TRUMPER, H. B., *Brit. J. Derm.*, 1933, XLV, 413.  
TELFORD, E., *Lancet*, 1937, I, 551.  
VAN SOMEREN, V., *Brit. Med. J.*, 1937, II, 1162  
WANTLAND, W., *Proc. Soc. Exp. Biol.*, New York, 1934-5, XXXII, 438  
WATSON JONES, R., and ROBERTS, R. E., *Brit. J. Surg.*, 1934, XXI, 461.

## PART THREE

### SECTION XI

## LOCALISATION OF FOREIGN BODIES

BY

T FANE TIERNEY MRCS LRCP DMR

### CHAPTER I

#### THE LOCALISATION OF FOREIGN BODIES

METHODS of localisation are exceeded in number only by their ingenuity and complexity. During the Great War numerous complicated X ray couches and pieces of apparatus were constructed with a view to making localisation a quick and easy measure. Under peace conditions and in civil hospitals and practice the call for these appliances does not arise and the localisation of foreign bodies is once more reduced to its fundamental principles.

If the foreign body is in an anatomical region where the full equipment and staff of the theatre is not required the X ray table may be made the operating table and the surgeon and radiologist thus co-operate. Periodic screen examination is thereby made possible and the progress of the operation checked. By this method a foreign body may be speedily extracted with consequent gain in time and minimal operative trauma.

Although it would appear on the first glance that two radiograms taken in two planes at right angles must necessarily indicate the exact position of the foreign body this is by no means true and right angled views may be regarded as an absolute guide only if the body is embedded in a piece of bone or if the two views are taken with the central ray passing through the foreign body and without moving the patient. Under the latter conditions the exact positioning of the part in question is essential.

The radiologist must always bear in mind that the problem is not a mathematical but an anatomical one and that some definite anatomical relationship is of much greater value to the surgeon than an exact physical measurement from a useless surgical skin marking. As an extreme illustration of this point information that a foreign body is lying in the knee joint is of great help whereas an exact mathematical description of a temporarily fixed object from an unmovable surface marking may be of little value.

The following methods are available and the selection of the method

depends on the site of the foreign body and the individual preference of the radiologist

### (1) METHODS OF DIRECT OBSERVATION

(a) **Fluoroscopy**—This method has the advantage of requiring no special apparatus and of being quick and in suitable regions exact. It is best carried out with the patient lying on the couch, the tube underneath and with the minimum size of diaphragm consistent with a true appreciation of anatomical relationship. The foreign body is first localised roughly and the diaphragm then contracted to a workable minimum. The region in question is now placed in such a position as can be easily reproduced and a mark made on the skin at the point where the shadow of the foreign body is cast by the central ray. The process is repeated in a plane at right angles to the first. The method of marking the skin is most accurately carried out by using a ring localiser, consisting of a small metallic ring carrying a pair of wires intersecting at right angles the whole mounted on a handle (Fig 548). A modification is to have a plunger which can be made to mark the skin at the point where the wires intersect.

The point of the intersection of the wires is placed over the foreign body under the screen which can then be removed the lights switched on and the point carefully marked with a silver nitrate stick or with Finzis ink (unless the type of ring used has its own marking arrangement). The main essential of this method is to record the exact position of the region under examination. If the surgeon decides to operate on the X-ray couch the first incision is now made and the part screened again as required. Pressure over the foreign body will cause movement of it and the nearer the forceps to the foreign body the greater the excursion it will undergo in this way greater control may be kept over



FIG 548 —  
Ring localiser

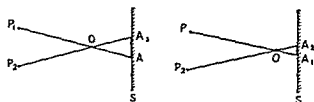


FIG 549—The parallax principle

the operation and any shifting of the foreign body will be immediately detected. A disadvantage of this method is the difficulty of keeping the operation field aseptic.

#### (b) The Parallax Principle

—If, as in Fig 549 a source of light,  $P_1$  is allowed to fall on to an object  $O$  placed in its path to a screen  $S$  a shadow of  $O$  will be cast at a point  $A_1$  on the screen. If  $P_1$  be now

moved to  $P_2$  another shadow will be seen at  $A_2$ . If now O be moved nearer to S the excursion of the shadow  $A_1-A_2$  is found to be markedly diminished. The magnitude of the excursion  $A_1-A_2$  may be taken as a measure of O from S providing the distance P-S remains constant. This is known as the *parallax principle* and may be utilised by replacing P by an X-ray tube and S by a fluorescent screen or film. O being the foreign body.

Use of this method may be made when the foreign body is removed on the X-ray couch. The relationship of the forceps to the foreign body is determined by moving the tube horizontally and adjusting the forceps until the excursions of both the foreign body and the forceps appear equal when this point is reached the forceps are approximately at the same depth as the foreign body from the screen and the only remaining factor is the lateral distance between the two shadows.

## (2) THE STEREOSCOPIC METHOD

Stereoscopic views are greatly favoured by many surgeons as they may give a good anatomical picture of the position of the foreign body. An essential for the success of the method is the presence in them of bony landmarks against which the foreign body must be orientated. In the absence of these e.g. the thigh stereoscopy is useless as an *exact* method of localisation. It is particularly useful in the skull. It is always wise to supplement the information gained stereoscopically by additional exact measurements obtained from one of the manipulation methods.

## (3) SIMILAR-TRIANGLE METHODS

It is by utilising the simple geometrical theorem of similar triangles that a large variety of methods have been evolved all depending on the consideration. Almost every radiologist has evolved some ingenious variant of the method and only the classical ones need be mentioned.

In Fig. 550 are two similar triangles.

Now if S represents the shadow shift on a screen or film F the foreign body and T the shift of an X-ray tube we may say that—

the depth of the foreign body D 
$$\frac{\text{tube distance} \times \text{shadow shift}}{\text{tube shift} + \text{shadow shift}} \quad \text{i.e.} \quad \frac{A \times S}{T + S}$$

We have now to consider how these conditions are applied.

The following measurements must be made

(1) Focus—film distance A (2) Tube shift distance T (3) Shadow shift distance S

For purposes of convenience (1) and (2) may be kept constant at distances easy for calculation e.g. 50 and 6 cm respectively.

Complicated apparatus is unnecessary for these methods a tape measure and plateholder with cross wires being all that are required.

There are three main methods

- (a) With a fluorescent screen
- (b) With one film and two exposures
- (c) With two films in cross wire holder

(a) **Fluoroscopic Method**—The part to be examined is placed conveniently on the couch and the tube screen distance arranged. Using a narrow pencil beam of X rays the position of the foreign body is first marked on the skin and then on the screen. The tube is now moved 6 cm horizontally the position of the foreign body again marked on the screen and the distance of the shadow shift measured. This by simple calculation gives the depth of the foreign body below the surface at the point already marked. Suppose the shadow shift was 2 cm. Then from the formula

$$D = \frac{A \times S}{T + S}$$

$$D = \frac{2 \times 50}{2 + 6} = \frac{100}{8} = 12.5 \text{ cm}$$

Various types of apparatus have been brought out with the object of making this measurement a simple matter one of the most satisfactory of which is that introduced by McGregor and used in the X ray units of the British Army.

THE MCGRIGOR FLUOROSCOPIC LOCALISER is designed particularly for use with a ward mobile unit and is independent of the focus screen distance. Attached to the tube is a cone bearing at its free end a lead diaphragm with an aperture corresponding to three quarters of a circle. The remaining quadrant projects into the circle with its point at the centre (Fig 551a). The ratio between the radius of the circular diaphragm and the focus diaphragm distance is 1 to 8.



FIG 551a—Diagram of the McGregor fluoroscopic localiser

**Fixed screen Method**—The localisation is simplicity itself. Patient and under couch tube are so adjusted that the shadows of the foreign body and the diaphragm pointer coincide (If the foreign body is large a prominent point in it is chosen for measurement purposes). This point is marked on the fluorescent screen. The tube is then moved till the shadow of the foreign body just disappears under the circular edge of the diaphragm. This point is also marked and the distance between the two points multiplied by eight equals the depth of the foreign body from the screen. As will be seen from Fig 551b this method is based on the ratio between similar triangles ABC and XYZ.

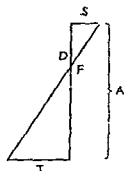


FIG 551b—Trigonometrical method

$A$  and  $A_1$  indicate the two positions of the focal spot and  $X$  the foreign body and as  $AC = SBC$  obviously  $XZ = SYZ$

It is convenient to have concentric circles each  $\frac{1}{8}$  inch apart marked on the screen. The depth can then be read off at once if the first shadow position is made to coincide with the centre of the circles. One disadvantage of the method is the penumbra cast by the diaphragm which may make accurate measurements difficult in thick subjects. It can be minimised by placing the cone as close to the under surface of the couch as possible.

**Moving screen Method**—This variation is used when the tube and fluorescent screen move synchronously. In this case the measurement must be made from a centre joint shadow instead of a screen mark or the circle centre point.

This is done as follows. Superimpose the localiser point and foreign body shadow as before and mark this point on the skin with a lead arrow. Now move the tube in a convenient direction till the shadow of the foreign body is cut off by the circular diaphragm. Mark this point of disappearance on the screen. Measure between this mark and the shadow of the arrow point as now visible. This distance multiplied by 8 gives the depth in inches as before.

(b) **Double exposure Film Method**—In this method the position is first localised

with a pencil beam of X rays and the skin marked. The diaphragm is now opened and a film placed over the area. A half exposure is given. The tube is now moved 6 cm and another half exposure given. On the film the shadow shift is measured and calculation is made exactly as in method (a).

(c) **Double film Method**—Where two films are used it is essential also to use a film holder with fine cross wires in order that there can be some fixed point from which to make the measurements.

The position of the foreign body may first be marked on the skin as before. A film is now placed in the holder the cross wires marked and placed in contact with the skin and an exposure made. The tube is shifted and another film exposed as before with the holder in the same position. It is well to get the two shadows in the same quadrant if possible and fairly near to the point of intersection of the cross wires.

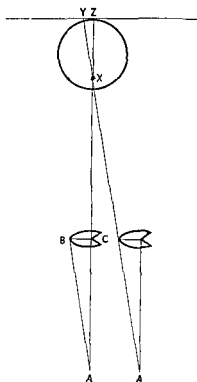


FIG. 51b. Diagram of the similar triangles in the McCorgor method.

The developed films are now superimposed by means of the cross wires the shadow shift measured and the depth of the foreign body calculated as before. If the horizontal position of the foreign body has not been ascertained, it may be found by using a simple paper modification of the MacKenzie Davidson localiser.

THE MACKENZIE DAVIDSON LOCALISER is now of historic interest only, but it illustrates visually the principles on which these methods are based and is worthy of description.

It is a simple piece of apparatus whereby the conditions used in taking the exposure are reproduced. Two threads are held by a cross arm over a base which has two lines ruled crossing at right angles. A tracing of the two films is made on a piece of tracing paper with the two positions of the foreign body indicated in relation to the cross wires. This is placed over the rulings on the base.

The two threads are now placed at the points of the shadow shift and held in position by small weights. The height of the cross arm is made to correspond to the target film distances and the threads are held at distances corresponding to the tube shift.

Thus the whole scheme of the exposure is reproduced. The point of intersection of the threads must correspond to the position of the foreign body. A perpendicular dropped from this point to the base will give its horizontal relationship to the cross wires and a measurement of this perpendicular distance will give its depth below the surface at this point.

*PART FOUR*

THE TEETH AND JAWS

By H. M. WORTH M.R.C.S. L.R.C.P., L.D.S.R.C.S., F.F.R. D.M.R.E.



## PART FOUR

### THE TEETH AND JAWS

By H. N. WORTH, MRCS, LRCP, LDSRCS, FFR, DMRE

#### CHAPTER LI

#### THE NORMAL TEETH AND JAWS

##### THE TEETH AND ALVEOLUS

UNLESS THERE is a clear understanding of the projection of shadows by X rays and a full appreciation of the normal appearances, the recognition of pathological conditions revealed radiographically is not possible.

It cannot be over emphasised that it is absolutely imperative to have a complete understanding of the normal appearances and their variations in order that departures from the normal can be recognised.

The Radiogram of a Tooth exhibits a white homogeneous shadow representing the dentine and cementum, which are not differentiated, and a whiter homogeneous shadow covering the coronal portion of the dentine and thinner at the neck of the tooth, representing the enamel.

The important point is that the enamel shadow is equally dense throughout. The pulp chamber and root canal are dark shadows in the centre of the tooth and larger in a young subject than in the adult. This is because there is a gradual diminution in the size of the cavity by deposition of dentine.

In a normal tooth the pulp chamber does not contain any evidence of structure.

The Periodontal Membrane, being a very thin soft tissue, does not cast any shadow but on account of its situation between bone and tooth both radio-opaque substances the space which it occupies is revealed as a thin dark line (Fig. 552). This is usually of equal thickness throughout its extent, but it is not unusual even in normal conditions to find an increased thickness of its cervical portion. The space is frequently termed the periodontal membrane, and this serves to save a lengthy description, but it must be understood that the actual membrane is not shown.

The thin dark line is seen to be continuous throughout its whole extent, but it may not appear to be of equal clarity and thickness on account of certain conditions of projection which will be discussed in describing the lamina dura.

The thickness of the membrane is not the same in every individual, but it is



FIG 552—Normal mandibular alveolus



FIG. 553—Normal maxillary alveolus



FIG 554—Normal maxillary bone of granular type



FIG 555—Normal mandibular bone



FIG 556—Absence of structure beneath the lower molars normal



FIG 557—Normal appearance suggesting the presence of a cyst



FIG 558—Anterior palatine foramen



FIG 559—Normal antrum



FIG 560—Normal antrum The grooves for the nutrient vessels are well shown



FIG 561—Lower incisors and geniohyoid tubercle



FIG 562—Upper incisors, nasal fossae and nasal septum



FIG 563—Normal developing tooth root

the same around each tooth in any one normal case. Any divergence from this is likely to be due to some abnormal cause.

The *Lamina Dura* is the thin cortical layer of bone which lines the tooth socket and which appears on a radiogram as a thin white line, usually, but not always quite sharply demarcated from the adjacent cancellous bone (Figs 555-556). Normally it is continuous throughout its whole extent, and can be traced around the roots, into the root bifurcation and up to the cervical surface of the alveolus.

It is an important structure in X ray diagnosis, and great stress is laid upon its continuity throughout its extent. From the above description it might be assumed that the lamina dura should be seen in every case as a sharply defined thin white line having equal density and width throughout its entire length. Such an assumption is incorrect and leads to considerable misunderstanding and error in interpretation.

Consideration of the projection of shadows and the exact anatomical shapes of tooth sockets will demonstrate that there must be considerable variation in the width, density, and sharpness of the shadow of the lamina dura in different cases.

The reason the lamina dura is seen in a radiogram and can be distinguished from the surrounding alveolus is that the bone of which it consists is denser and therefore more radio opaque than the surrounding structures.

The rays passing through the alveolus enter the cortex of the socket and experience the greatest absorption when the angle of incidence is in the long axis of the longest length of the condensed bone, namely at the mesial and distal aspects of the tooth. The greatest absorption and therefore the sharpest shadow will result when the walls of the socket are flat, and parallel mesially and distally. If, however, as is very frequently the case, the shape of the root is irregular there will not be a great length of socket cortex to absorb the beam of rays, and the resulting shadow will not be dense nor sharply defined. Furthermore on account of the concavity or convexity of the walls of the socket, the total width of the projected shadow will be greater and consequently less sharply defined. This explains what is often a cause of concern to many interested in dental radiography, and it can be taken as an example of the necessity to keep the simple facts of shadow production constantly before one.

The Alveolus which surrounds the teeth and acts as their support may present a variety of radiographic appearances (Figs 552-554). In general they can be described as resembling a network pattern, usually more closely set in the maxilla than in the mandible.

In the maxilla there is a normal variation in different individuals—from a more or less granular appearance (Fig 554) to a fine network (Fig 553) but the pattern is the same throughout in any one person. In the mandible there is a wider variation extending from a few irregularly placed intersecting trabeculae in the infant through a gradation of wide, less wide and small mesh

network in the adult (Figs 557 and 558). The granular appearance sometimes seen in the maxilla is not encountered in the mandible.

It is not uncommon in adults to find no network pattern but a few trabeculae which may be arranged so as to simulate the walls of a cystic cavity. Such cases may be operated upon in the belief that they are pathological (Figs 556, 557). The knowledge that such an appearance may be normally encountered should make the observer very cautious of diagnosing the condition as cystic or pathological in the absence of clinical evidence.

In the mandible the network appearance is frequently everywhere present except beneath the roots of the molars and premolars there being no structure other than the inferior dental canal between these teeth and the inferior border of the mandible (Figs 556 and 564).

This absence of structure is also a common source of difficulty especially when associated with clinical evidence of some abnormality. The condition has been diagnosed as neoplasm, necrosis, and cyst, and some cases have been subjected to surgery. In quite a number of persons the network is continuous throughout the whole extent of the mandible with very little variation.

In the molar and premolar regions the alveolus between the teeth forms flat tables known as interdental tables and situated at the level of the necks of the teeth. Very rarely the surface of the table is corticated and this is revealed as a thin dense white line at the margin of the bone. It is much more common, however, for the surface of the alveolus to be clearly defined but with no evidence of a cortex and this must be regarded as the radiographic normal. The interdental table makes a sharply defined angle with the lamina dura (Fig. 552).

In the incisor region where the teeth are less bulbous the alveolus forms spines more or less sharp depending upon the closeness with which the teeth are set.

In the lower incisor region sometimes quite sharp points are present produced by the approximation of the laminae durae of adjacent teeth which are very close to one another.

## THE MANDIBLE

The Inferior Border of the mandible has a thick cortex which casts a homogeneous light shadow like a band bordering the bone about one-eighth inch wide. It is usually absent behind and commences just in front of the irregular portion of the inferior surface of the angle—that is in front of the insertion of the masseter becoming thinned out and lost in the incisor region (Fig. 564).

The Inferior Dental Canal may be very clearly or only faintly seen. At its clearest it is revealed as two crescentic white lines concave upwards and slightly forwards which commence in the upper portion of the ascending ramus as a dark area over which the lingula may or may not be shown. Passing

forward beneath the roots of the molars and premolars, it usually ends at the mental foramen, but occasionally the canal is seen to continue forward beneath the canine, in which case there is a small projection of the canal upwards beneath the premolars and ending in the mental foramen (Fig. 564). This appearance is sometimes a cause of concern to the uninitiated.

The shadow of the inferior dental canal may be superimposed over the apices of the last two molars, suggesting that the roots may be in the canal, but the continuity of the structure indicates that the tooth is outside.

In the presence of superimposition of the molar roots and canal it is sometimes observed that the periodontal membranes seen through the canal appear to be thickened. This is a projection effect and must be kept in mind.

The Mental Foramen is not always seen, but it may be very clearly revealed as a dark, well-defined more or less circular area situated below and usually between the roots of the two premolars (Fig. 564). Quite often it is represented by a small dark area not very clearly demonstrated. The shadow is occasionally superimposed over the apex of one of the premolars, usually the second one, and may be mistaken for apical infection. The differentiation is made by the presence of an intact lamina dura in the absence of disease.



Fig. 564.—Radiogram of a dried mandible.

The Internal and External Oblique Ridges may be superimposed over one another or their shadows may be cast separately. They appear as dense white lines, the external one being usually more anteriorly placed, and both pass downwards and forwards, covering the cervical portion of the third molar and gradually merging imperceptibly with the body of the one (Fig. 564).

The Genio-hyoid Tubercle is nearly always seen in a radiogram as a small round white opacity with a central dark spot. It is situated in the midline of the mandible well below the incisor teeth (Fig. 561).

The Coronoid and Condylar Processes are also shown in appropriate radiograms.

There is sometimes an appearance in the sigmoid notch which may be mistaken for a pathological condition. It is a dark area situated in or near the most concave portion of the notch, and is due to a small fossa or recess which

occurs anatomically in some individuals. It has no clinical significance except that it has been mistaken for a pathological lesion.

There are other appearances which are sometimes found in radiograms of the lower incisor region and which are due to anatomical causes but may be mistaken for pathological conditions.

One of these is the presence of dark lines commencing in the alveolus between the incisors and less commonly the premolars and running down parallel with the teeth towards the body of the jaw where they gradually terminate.

The other appearance is due to a marked concavity which is sometimes found in the anterior surface of the alveolus and body of the mandible in the incisor region which on account of the lessened density of bone results in a dark area in the radiogram which may be mistaken for an area of bone rarefaction.

Other structures which must be noted in a description of X ray appearances of the mandible are the stylo-mandibular ligament, which occasionally ossifies and the hyoid bone which is frequently superimposed on the mandible in extra oral radiograms.

## THE MAXILLA

The Inter-maxillary Suture appears as a dark line separating the two maxillæ (Fig. 562). Its position is absolutely central and this together with the fact that there is a thin cortex to each of its bony borders renders the differentiation from a fracture easy. It is said to commence to fuse from behind forwards about the age of 42.

The Anterior Palatine Foramen is a dark area situated in the premaxilla with those portions on each side of the midline almost symmetrical. It may appear well above the roots of the incisors between them or at the apices of these teeth. In the latter position difficulty may be experienced in ascertaining that there is no pathological condition present (Fig. 568).

The Incisive Fossæ may appear as slightly dark areas situated over the lateral incisor regions and may be mistaken for bony disease. The absence of any bony absorption as indicated by loss of structure taken in conjunction with the symmetry of the condition usually render its recognition easy.

The Nasal Fossæ appear as large structureless areas situated above the root of the incisors (Fig. 562).

In children it may appear that the roots of the incisors are actually in the fossæ but this is due to projection of the shadows and is not an accurate anatomical relationship. In adults the fossæ are usually well above the roots of the teeth but confusion may arise in certain cases if the presence of the shadows is not understood.

The Nasal Septum is revealed as a white line dividing the shadow into two halves and the lower border of the fossæ is seen to be lined by a definite bony cortex (Fig. 562).

Sometimes the nasal spine is seen as two white lines coming to a sharp point in the midline above the incisor teeth

It is not uncommon for a shadow to be cast by the soft tissue of the nose, and for the nasal openings to appear as symmetrical and well defined dark areas. They have been mistaken for areas of bone rarefaction.

The Maxillary Antrum appears as a dark cavity usually extending from the first premolar to the third molar region and having a thin white cortical margin.

It has a great variety of shapes and sizes and considerable care has to be used in some cases in differentiating a normal antral shadow from that of a cystic cavity (Figs 559-560).

It is by no means an infrequent occurrence for a normal antrum to be opened surgically under the impression that the dark shadow revealed in a radiogram is due to some pathological condition. Thus mistake is not likely to arise if the normal variations are clearly understood and if the clinical evidence is given its proper importance.

Usually the floor of the antrum is situated above the roots of the teeth but occasionally it is seen to dip down between the teeth to the alveolar margin. In edentulous patients the floor of the antrum may be formed by the alveolar border (Fig 560). It is in such cases that errors of interpretation are likely to occur and in the latter type the removal of a small piece of root is fraught with the real danger of opening the antrum.

The antral cavity sometimes appears to be divided by bony partitions, which may be vertical curved or spherical and in the last case it may be quite impossible from radiographic evidence alone to exclude a pathological condition (Figs 559-560). There are also present in the antral shadows thin dark grooves which are produced by the vessels and nerves which pass over the walls of the sinus (Fig 560). The roots of the teeth may appear to project into the antral cavity but it can be taken as certain that this does not occur in a normal case. The amount of bone separating a root from the antrum may be very thin but it is always present in the absence of disease or trauma. This fact is clearly proved by the presence of the lamina dura which indicates the presence of a complete bony covering to the tooth root.

On account of the angle at which the X ray must strike the antrum and teeth in order to produce a satisfactory radiogram there are many films which suggest that the roots and the antrum are very closely related whereas anatomically this may not be the case.

The Malar Bone is very frequently seen in an intra-oral radiogram of the maxillary molars. In those cases in which the palate is flat or the cheek prominent and low the shadow will be most obvious and most likely to obscure the roots of the teeth. In many cases it is not possible to avoid the superimposition of the shadows of the malar bone over the roots of the molar teeth and as a result the diagnostic value of the radiogram is reduced.

On account of the shape of the malar bone and the projection of the antral

cavity into it there is a white sharply curved appearance produced in a radiogram by that portion of it which is in continuity with the maxilla, whereas the zygomatic portion appears as a more or less even opacity of variable density.

The zygomatico malar suture may appear in an extra-oral radiogram of the maxilla and may simulate a fracture.

The Coronoid Process of the mandible is sometimes seen in an intra-oral radiogram of the maxillary molar region and may be mistaken for a tooth but the absence of tooth structure and the lesser density of the coronoid process together with its position make the observer suspicious and confirmation may be obtained by repeating the radiogram with the mouth widely open when the shadow of the coronoid process will disappear.

The Hamular Process of the pterygoid bone occasionally appears in extra-oral radiograms taken sufficiently far back. In occlusal views of the maxilla there is a round dark area seen in the molar region which is commonly interpreted as being the posterior palatine foramen but it is really a shadow of the infra orbital foramen.

### VARIATIONS OF ROOTS

Variations in the number of roots occur and can usually be recognised in a radiogram but there are occasions in which additional apices do not appear and cannot be seen even when known to be present.

The third molar is the tooth usually affected.

Roots which bend sharply backwards are not infrequently found and the state of affairs can be seen in a radiogram. When the bend is in the buccal or lingual direction the presence of a small white ring of lamina dura around a dark one of periodontal membrane enclosing a slightly denser area of tooth substance situated at the apex indicates that the root is bent. It is not possible to ascertain whether the deflection is a buccal or lingual one.

### THE TEETH AND JAWS IN CHILDREN

There are certain differences in the appearances of normal jaws of children as compared with those of the adult.

The structure of the bone is very much less marked and in place of a net-like appearance there may be a few irregularly placed trabeculae intersecting one another at wide intervals. In other cases there is no structure to be seen in the body of the bone of the mandible except at the inferior border. In the maxilla the greater part of the alveolus is occupied by the unerupted teeth which obscure the bone structure.

The unerupted teeth in their crypts are situated beneath the deciduous teeth or in the case of the molars in the bone behind them and the depth at which they are situated depends upon the age of the patient and the nearness of the time for eruption. The crypts themselves are sharply defined spherical dark areas with well marked cortical linings represented by thin white

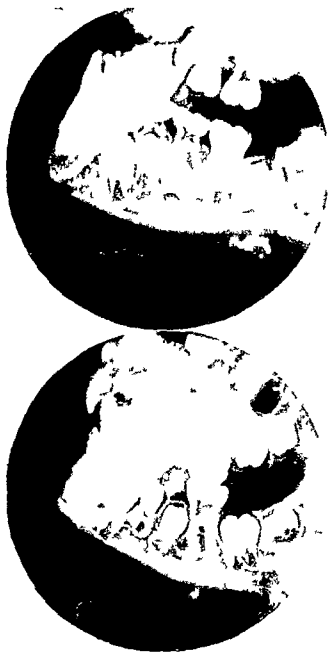


interrupted white lines (Figs 565-566)

The partially formed teeth are situated inside their crypts and are separated from the walls by a small space

The crypts of the developing teeth may be present, but with no evidence of tooth substance inside. As the process of eruption commences the alveolar aspect of the crypt becomes absorbed in readiness for the emergence of the tooth. Any loss of continuity of the cortex lining the crypt other than that which occurs in preparation for eruption is likely to be due to infection. Other appearances are similar in adult and child. The apices of teeth which have not finished their growth have characteristic appearances: the apical end of the root canal is open and funnel shaped so that it is the widest part of the canal (Fig 563). This is in contradistinction to the apex of a tooth which has completed its growth and later become absorbed when the pulp canal is narrowest at the apical end.

The lamina dura extends to the extreme end of the portion of the root which has formed, and even the smallest departure from this is usually indicative of a septic condition at the apex.



FIGS 565-566.—Radiograms of normal mandibles in children

## CHAPTER LII

### UNERUPTED AND SUPERNUMERARY TEETH

MUCH USEFUL information may be obtained in the diagnosis of the presence of unerupted teeth and in their localisation by means of careful radiographic study.

In orthodontic work especially the X rays must be constantly used both as a means of ascertaining the cause of irregularity resulting from unerupted teeth and in estimating progress.

Apart from the recognition of unerupted teeth their position can be ascertained by carefully made radiograms taken in two or more planes and in those cases in which the tooth is completely buried occlusal views are very valuable in order to reveal its relation to the dental arch (Fig 568). It is inadvisable to attempt the removal of completely buried teeth with only one radiogram taken in one plane and it may be regarded as generally true that where it is possible for an intra-oral radiogram to be obtained it is more informative than an extra oral one. Stereoscopic views are valuable in certain cases but are not so valuable as radiograms taken in several planes.

Having obtained radiograms with the minimum amount of distortion these are studied in order to ascertain the presence of the tooth its position its relation to other teeth and structures such as the inferior dental canal and maxillary antrum the number direction and shape of the roots the presence of any pathological condition or other abnormality. There are certain parts of the jaws which are more commonly the site of unerupted teeth and they will be considered separately.

*The upper incisor region is the commonest site of unerupted supernumerary teeth which may or may not be causing delayed eruption of the permanent teeth or displacement of erupted ones. Supernumerary teeth may be single or multiple and are occasionally encountered in an inverted position (Fig 569). When single they are usually found in the midline of the maxilla but when multiple they occur on each side of the midline and usually behind the permanent teeth. In those cases in which there are unerupted permanent and supernumerary teeth it is sometimes very difficult to unravel the group of shadows. The supernumerary teeth are usually recognised by the fact that their crowns present an irregular appearance but there are a few cases in which it is impossible on radiographic evidence alone to decide the exact state of affairs. The upper lateral incisors are frequently absent and this fact has to be ascertained in many cases before treatment can be commenced. When they are present it is not difficult to recognise them. The upper*

canine is very seldom absent, but cases do occur in which this tooth does not develop. Of all the upper teeth the canine is probably the one most commonly radiographed in order to ascertain its position. In a young child it is situated very high and apparently out of place, but considerable caution is necessary in attempting to decide whether it will



FIG. 567.—Unerupted canine teeth.



FIG. 568.—Occlusal view of unerupted tooth situated palatally.



FIG. 569.—Inverted supernumerary tooth.



FIG. 570.—Unerupted lower teeth.



FIG. 571.—Geminated upper incisor.

erupt normally. This tooth has frequently a forward inclination of the crown which suggests that it will be displaced, but with the natural changes which occur during growth of the bone the tooth eventually assumes its normal position. There is a tendency for the unexperienced observer to be misled into thinking that canines and premolars in a normal child are crowded, but it must be remembered that the growth of the bone results in normal eruption in most cases.

The canine is very frequently displaced (Figs 567 and 570) either slightly or grossly and the lateral incisor may be rotated or tilted by pressure on its root by such a tooth this can only be accurately decided by radiograms obtained from different positions. The canines may be situated obliquely or horizontally and come into close relationship coronally with the posterior aspects of the central incisors. In other patients the tooth is situated across the dental arch so that one end of it presents towards the buccal sulcus and the other in the palate. This position should be recognised before any operative interference is commenced because it may lead to considerable difficulty in extraction with injury to other teeth. The upper canine may occupy positions other than those recognised the same points arise in all of them.

Occasionally it is found to be situated immediately below and in very close contact with the antral or nasal cavities.

Notwithstanding the most careful radiographic study cases will be encountered in which the evidence afforded has been somewhat misleading. In edentulous cases the unerupted canine is not infrequently encountered long after all the other teeth have been removed and may be found to be associated with bone destruction around its crown.

In all unerupted teeth especially where sepsis is present there is a tendency for absorption to occur which does not resemble the appearance of caries and which may be so extreme as to render the recognition of the tooth very difficult.

The upper premolars develop in a manner which suggests that crowning is present with the second premolar above the first one. This condition may persist so that the tooth remains high up or is displaced to the buccal or palatal side of the dental arch. The upper first and second molars are not commonly misplaced or unerupted but either may be prevented from erupting by impaction. The upper third molar is frequently unerupted with or without impaction and it is commonly found with the crown facing backwards occasionally the tooth is inverted so that the apex presents on the alveolar border.

In the mandible it is much less common to find supernumerary teeth and when they do occur it is usually in the premolar region. The incisors are sometimes absent but the canines are not nearly so frequently found to be displaced as the upper ones. The premolars are sometimes absent and the second one is found to be the one most commonly unerupted in adults. In such cases the tooth may be impacted between the adjacent teeth or completely displaced from the arch. The lower third molar is very often the cause of symptoms demanding its removal on account of displacement from its normal position. In any case where an unerupted third molar is present carefully prepared radiograms are needed in order to furnish the most complete information so that the operation for its removal may be facilitated. Intra oral radiograms offer the greatest evidence and more accuracy but they

are not always obtainable. The extra oral film should only be used when it is impossible to obtain intra oral ones.

The rays must be directed in such a way that they pass between the crowns of the first and second molars so as to define clearly the contact points without superimposition. This is one of the criteria of the correctly posed radiogram. The second criterion is that the buccal and lingual cusps must be accurately superimposed indicating that the rays have passed through the plane of the occlusal surface.

If the above details are given their full significance there will be no misrepresentation of the true state of affairs and it will be possible to estimate the degree of impaction of the unerupted third molar, the amount of bone over the tooth and the presence of erosion of the posterior surface of the second molar (Figs 572-575). Other

information which is sought is the condition

tion of the tooth itself and of the adjacent teeth, the number, position and shape of the roots of the unerupted tooth, the presence of any space beneath or beside the crown which may be of value in removing the tooth, the relation of other structures such as the inferior dental canal, the oblique ridges, the ascending ramus and also the presence of any pathological condition in relation to the tooth. In vertical impaction there is frequently a pocket in the bone behind the crown which may be of considerable importance in the treatment of the tooth. In those cases in which the tooth is completely buried a radiogram should be taken in



FIG 572-574 — Unerupted impacted lower third molars



FIG 575 — Unerupted lower third molar situated across the arch



FIG 576 — Occlusal view of unerupted third molar

the occlusal plane to reveal any deviation of the tooth toward the inner or, more rarely, the outer side of the dental arch (Fig. 576)

**Supplemental Teeth** are most commonly found in the upper lateral incisor region, and they appear as extra teeth almost identical in shape and size with their fellows. Supplemental teeth are occasionally found in the lower premolar region and more rarely in the molar area.

**Dilacerated Teeth** are usually found in the incisor region and are readily recognised as such in a radiogram.

**Geminated Teeth** are usually identified radiographically (Fig. 571), but there are some cases in which it may not be possible to decide whether the teeth are really fused.

## CHAPTER LIII

### INFLAMMATORY DISEASES OF THE PERIODONTAL MEMBRANE AND ALVEOLUS

#### PERIODONTAL DISEASE (PYORRHŒA, PARADONTAL DISEASE)

THE PATHOLOGY of the above condition is said to be that of a progressive marginal gingivitis associated with rarefying osteitis resulting in alveolar absorption, and it is stated that the rate of progress of the disease is closely related to the density of the bone and the functional activity of the teeth

*Radiographic appearances appear to support these statements*

All cases in which there is alveolar absorption must not be considered to be abnormal because as the age of the subject increases there is a tendency for the alveolar margin to recede. In a radiogram of the normal subject the surface of the alveolar border is flat between the molars and premolars and more or less pointed between the anterior teeth depending upon the closeness with which the teeth are set

The surface of the alveolar bone is sometimes corticated but this is an unfrequent finding and it is far more common to see no evidence of a cortex but the density of the bone the same throughout

In the absence of any demonstrable disease the periodontal membrane at the cervical margin may appear to be thickened, and this must be regarded as a normal variation. In other regions the periodontal membrane is of equal thickness. The changes which are seen depend upon the virulence of the infection and the length of time during which it has been active, and also upon the resistance and age of the subject

There are numerous different appearances revealed which have not equal significance in every case, so that it is necessary that every aspect must be studied in arriving at a definite conclusion (Figs 577-584). In examining a radiogram, the first thing that is studied is the position of the alveolar border relative to the root of the teeth

In periodontal disease there is a destruction of the alveolar border, resulting in the recession of the alveolar margin towards the tooth apex. The fact that there occurs a non pathological recession of the alveolar border in elderly people must always be considered and estimated in attempting to arrive at a correct appraisal of the true state of affairs. This offers considerable difficulty in some cases. Whereas in early cases of periodontal disease the level of the alveolus may have receded but slightly, in advanced conditions the whole of the tooth roots are completely denuded of bone. Marked alveolar absorption



FIG 577—Alveolar absorption on diseased quiescent



FIGS 578-579 Marked alveolar absorption without rarefaction of the alveolar margin



FIG 580—Alveolar absorption with marked marginal rarefaction



FIG 581—Advanced alveolar absorption with healthy bony margin



FIG 582—Pocket formation



FIG 583—Deep pocket formation



FIG 584—Pocket formation localised to one tooth



FIG 585—Alveolar absorption extending below the root surface

in a young subject is of more importance than a similar amount in an older person whereas an extensive amount of recession is likely to be a serious finding in any individual

It is apparent therefore that there are encountered many cases in which the alveolar absorption is such that an accurate estimate of its significance is not possible except by careful clinical consideration

The level of the alveolus tends in some cases to remain flat while in others



the bone is more markedly destroyed immediately adjacent to the tooth thus resulting in pocket formation

Pocket formation is more serious than alveolar absorption of the horizontal type. The absorption is sometimes seen to affect the buccal or lingual aspect only or more severely and unless careful study is made this may be overlooked. It is recognised in the radiograms by the presence of a line of different density passing across the roots at a lower level than the major portion of the alveolus. It is important both on account of the fact that it may be easily overlooked and also because it may result in the formation of foci of infection. The bone between the bifurcation of roots is frequently completely destroyed in alveolar disease and when it is marked the condition is obvious. In very early stages however the root bifurcation may be slightly involved and this is recognised by the presence of a small dark area of bone rarefaction at the extreme apex of the alveolar septum (Fig 385). Such areas of rarefaction hidden beneath the gum are possible foci of infection and on account of their position and the slight amount of evidence which they reveal they are of considerable importance. It has been noted that the inter dental table (that is the surface of the alveolus between adjacent teeth) forms a clearly defined angle with the lamina dura and this is a state of affairs which may still be present with any degree of alveolar absorption.

In other cases this angle is commonly seen to be destroyed so that a pocket is found at the neck of the tooth. The presence of such a condition is less favourable than is the case when the angle is maintained.

There is another very important manifestation to be considered and that is the presence or absence of rarefaction of the alveolus. In some cases the alveolus while it is obviously absorbed does not reveal any change in the density of the bone (Figs 577-579) but in other individuals there is rarefaction involving the alveolus to a greater or lesser extent. Rarefaction is shown by the presence of a darker shadow in the radiogram and it is seen to be due to osteoclasia of bony trabeculae with loss of continuity of some of these structures (Fig 580). The bone spaces on the surface of the alveolus may be open superficially.

Rarefaction of the bone indicates the presence of a more important state of affairs and the greater the depth of bone which is involved the more serious is the infection and less good is the reaction considered to be. The presence of slight alveolar absorption with rarefaction is more serious than a greater amount of absorption with no rarefaction.

On the other hand, cases with any degree of alveolar absorption unassociated with loss of the angle of the inter dental table and lamina dura, and presenting a clearly defined dense surface, are probably the result of non pathological absorption or of healed periodontal disease (Fig 577). The condition of the periodontal membrane as revealed by its thickness (width) must be considered. Many cases of periodontal disease do not reveal any evidence of

change in the width of the membrane and this is usually regarded as being a favourable indication but there are encountered other cases in which there is thickening of the membrane as a whole or of that part immediately adjacent to the alveolar surface. The degree of involvement of the membrane is usually regarded as being one indication of the seriousness of the disease. The level of the gum margin is imperfectly revealed in a radiogram but it must be considered because it is of importance in estimating the presence of foci of infection. For instance a patient presenting marked alveolar absorption with little or no recession of the gum is considered to be in a less favourable condition than one in which the gum has maintained a normal position relative to the alveolus. The reason for this is that the space which exists between the gum and the bone in the former case offers an excellent place for bacteria to multiply and produce their toxins. In patients with marked pocket formation the gum stands above the level of the highest portion of alveolus and consequently spaces are formed offering a splendid harbour for bacteria.

From the above it will be seen that the consideration of periodontal disease is not complete when based on radiographic evidence alone. The condition revealed on clinical examination taken in association with the radiographic evidence the age and the health of the patient must all be considered but notwithstanding this it is very common for radiologists to be asked to recommend treatment on their evidence alone.

### APICAL INFECTION

Apical infection may result in changes in the root or in the bone surrounding the root or in both of the structures at the same time. Bony changes are of an inflammatory nature and result in rarefaction or sclerosis or a combination of each. Rarefying osteitis is the term given to those conditions which have been named chronic abscess, peri apical necrosis, apical granuloma and alveolar abscess. Rarefying osteitis may be acute or chronic.

**Acute Rarefying Osteitis**—In acute infections there may be no radiographic evidence of abnormality even in the presence of marked clinical manifestations. This applies to all acute bone infections and as many as ten days may elapse after the inception of the disease before there are sufficient changes in the bone to become recognisable in the radiogram.

It is not possible to state definitely how long it will take for osteitis to reveal itself in any individual case. In those cases in which a chronic infection has become acute the appearances are usually those of the chronic condition at least in the early stages. In acute alveolar abscess or acute rarefying osteitis the earliest changes are those of rarefaction of the bone at the apex of the affected tooth associated with a breach in the continuity of the lamina dura and perhaps thickening of the periodontal membrane at the apex. The rarefaction reveals itself as a dark area.



FIG. 583.—Large area of apical osteitis local sequestra corticated

FIG. 587.—Apical osteitis local sequestra

FIG. 585.—Diffuse area of apical osteitis



FIG. 589.—Apical osteitis with well defined cortex



FIGS. 590 and 591.—Small areas of apical osteitis



FIGS. 592 and 593.—Small areas of apical osteitis

FIG. 594.—Osteitis at apex of second premolar with new bone at antral floor

Later the trabeculae may be completely destroyed in the affected area and the adjacent edges of bone become rarefied and irregular.

Associated with this there is definite destruction of some part of the apical portion of the lamina dura. In the absence of the latter it is not possible to diagnose radiographically the presence of apical osteitis—acute or chronic—and this is a point of differentiation from other conditions which do not involve the tooth over which the unusual shadow is projected. In all cases of osteitis which can be recognised in a radiogram there is a breach in the continuity of the lamina dura if the tooth is involved in the disease process and with very rare exceptions the affected tooth is a dead one.

In acute apical infection the disease may spread to involve adjacent teeth and where there is doubt as to whether this has occurred the involvement of the lamina dura as revealed by a breach in its continuity is the deciding factor.

**Chronic Rarefying Osteitis**—Chronic rarefying osteitis may simulate the appearance of the acute condition but in the former the areas of bone destruction tend to be more clearly defined. It may however not be possible to differentiate the two types on radiographic evidence alone.

There is considerable variation in the degree of bony involvement ranging from small localised areas to large diffuse ones (Figs 586-593). Areas of bone destruction of any size may show a sharp well defined border of demarcation (Figs 586-587) or they may be diffuse and very indistinctly delimited from the surrounding bone (Fig 588). In all cases in which there is definite bone destruction it is revealed in a radiogram as a dark area at the apex of a tooth or teeth which indicates the extent over which the bone has been destroyed.

In some cases the area of bone involvement is surrounded by a thin white bony cortex (Fig 589) in others the diseased area merges imperceptibly with the adjacent bone. It used to be taught that the diffuse ill-defined areas were due to granuloma and the clearly defined corticated type was associated with so called chronic abscess but it is now known that it is not possible to differentiate the two conditions on radiographic evidence. It is generally accepted that all types are infective and that the presence of a cortex does not indicate sterility that the diffuse localised and corticated types may all contain organisms of a virulent nature and all that can be inferred from the various appearances is that the reaction on the part of the patient is more marked in localised areas than in diffuse ones. The accuracy of this view has been clearly established by bacteriological evidence. In diffuse rarefying osteitis adjacent teeth may be involved by direct spread (Fig 589) but it is not unusual for an area of bone destruction to appear to involve the next tooth when this is not really the case. Study of the radiograms will show the lamina dura of the suspected tooth to be intact and the tooth therefore to be uninvolved.

There are occasions when doubt exists as to whether a dark area at the apex is due to bone destruction or to superimposition of a foramen and it may be necessary to resort to tests of vitality in order to decide the point.

It can be accepted that except in very rare instances the presence of bone destruction at the apex of the tooth and involving the tooth implies that the tooth is dead. In those cases in which the bone rarefaction is not well marked the presence of abnormality may be overlooked unless great care is taken in the examination of the radiogram especially of the lamina dura. A very small dark area over the root apex which on close examination is seen to be due to slight destruction of the bony trabeculae and associated with a breach of continuity in the lamina dura is all the evidence which is revealed in many cases of apical infection. In some positions where there

is normally found an anatomical excavation of the bone, such as the incisive fossae the mistake of regarding such an appearance as osteitis is avoided by noting that the cortex of the tooth socket is quite intact.

In certain situations the recognition of the presence of apical osteitis is rendered more difficult such being anatomical proximity of the apices of certain teeth to the floor of the antrum and the superimposition of the antral shadow over the roots of the molar teeth. In these cases in which the roots of the teeth are situated immediately beneath or very close to the antral floor there is very little bone at the apex to be destroyed so that the osteitis causes the floor of the antrum to become absorbed over the involved



FIG. 591—Osteitis at apex of palatal root with the antrum superimposed.



FIG. 592—Thickened menbranes at the apices of the first molar.



FIG. 593—Slightly thickened membrane at the apex of the first premolar.



FIG. 594—Slightly thickened membrane at the apex of second premolar with periapical sclerosis.



FIG. 595—Sclerosing osteitis at the apices of the molars.



FIG. 600—Diffuse sclerosing osteitis.

areas and the tooth root may be in direct relation with the antral cavity. This is a rare event and usually the antral cavity is kept intact by the action of the osteogenic layer of the mucoperiosteum lining the cavity, which lays down new bone over the infected area. In a radiogram of such a condition the tooth apex is seen to be denuded of bone, and the resulting dark space surrounded by a thin layer of bone which in appearance somewhat resembles a 'halo' and which is continuous at the sides of the root with the lamina dura (Fig. 590). This appearance is seen at the apices of the premolars and buccal roots of the molars. The fact that the large dark space of the antrum is immediately adjacent to the roots of these teeth sometimes tends to cause

these appearances to be overlooked. In a similar manner the palatal roots of the upper molars are often seen through a dark antral cavity so that any small areas of infection at the apices of these roots are more likely to be overlooked and as there are normal variations in the density of the shadow cast by a normal antrum this likelihood is accentuated when such variations are superimposed over a tooth apex (Fig. 595).

The diagnostic point is the presence or absence of continuity of the lamina dura. The smallest change indicative of apical osteitis is seen to be a thickening of the periodontal membrane localised to the apex only for in order for this to occur there must be absorption either of the tooth or bone (Figs. 596-597).

When it is the bone which is absorbed the lamina dura will be seen to be thin but probably not completely destroyed over the involved area and this indicates the presence of osteitis. Further evidence of the presence of infection may be the association of sclerosing osteitis outside the thickened apical periodontal membrane (Fig. 598). There is one condition which may be confused with this appearance—the normal one seen when roots of a lower molar tooth are superimposed over the shadow of the inferior dental canal. By an effect of projection the periodontal membrane appears to be thickened at the apices in such cases but it is not a pathological condition.

All the types described so far have been at the apices of the teeth but there are occasions in which bone destruction is seen at the sides of the root. In many of such cases there is a perforation of the root by instrumentation but there are some in which there has been no treatment and the probable explanation is that the infection has commenced at one of the arborisations of the pulp canal instead of at the extreme apical foramen.

Occasionally there will be seen to be obvious osteitis present with a very much less distinct lamina dura in the involved area. The explanation of this is that the disease process is situated in such a position that only one segment of the root is involved and the lamina dura is seen in profile through the rarefied area (Fig. 593).

**Sclerosing Osteitis.**—Sclerosing osteitis is encountered in a variety of different conditions. Its most common occurrence is at the margins of an area of bone destruction at the tooth apex where it may be seen as a thin line forming a cortex or as a more diffuse condensation with no sharp line of demarcation from the healthy surrounding bone (Fig. 599). It is frequently found at the apices of teeth without any bone destruction where it may be slight in extent and not very dense or it may extend considerably away from the tooth roots (Figs. 599-600).

The types described above are due to infection of the bone and represent a reaction to infection. *Fish* has demonstrated by means of his histological sections of post mortem specimens that these areas are quite definitely infected. Occasionally there are revealed in radiograms dense areas of bone sclerosis situated some distance from the teeth not obviously related to them.

and with no evidence of any oral sepsis to account for their presence. Early writers named such areas when small *bone ichorls* but offered no explanation of their origin. Nothing appears to have been added to our knowledge of this type of bone involvement and it is customary to regard the condition as non-pathological.

Tooth sockets sometimes heal and fill with dense sclerosed bone which is only differentiated from a tooth root by the absence of spaces for periodontal membrane and root canal.

While these criteria usually serve to distinguish the two there are occasions when the presence of sclerosing osteitis around the socket which contains the root renders the differentiation impossible. From the clinical point of view sclerosing osteitis due to sepsis is regarded as a possible focus of infection and measures are sometimes taken to excise the involved area. It appears to be usual however to regard the condition as a localising one and not to interfere except in cases of neuralgia when this is thought to be caused by pressure on nerve endings or on the inferior dental nerve in the socket or massive types respectively.

The bosses of bone which are sometimes found on the inner side of the mandible in the premolar region and the so called *torus palatinus* of the maxilla do not contain sclerosed bone and do not therefore usually reveal any evidence of their appearance in a radiogram. The radiographic appearance of sclerosing osteitis is quite typical for in the place of the usual pattern the bone spaces are filled in with new bone producing a shadow of homogeneous density.

Colyer describes the condition in earlier stages as showing a circular appearance of the bone spaces due to a deposition of bone on their inner walls. Sclerosing osteitis has to be differentiated from a buried root, from productive periodontitis and from a solid type of odontome. The last is more dense in appearance and shows the presence of a capsule which separates it from the bone whereas in the case of sclerosing osteitis there is obvious continuity with the surrounding bone. An ivory exostosis may not be distinguishable except by stereoscopic radiograms when it will be seen to be on the surface of the bone. Usually its shadow is denser than that of sclerosing osteitis.

**Residual Sepsis**—Residual sepsis in edentulous areas is recognised by the presence of a number of radiographic appearances (Figs 601–606).

The term implies that infection which was present in the alveolus when teeth were present has persisted after their removal, it is also used to include infection in the tooth sockets which were not infected previous to extraction.

Radiographically the appearances are varied and call for considerable care in appraisal. Full consideration of the length of time which has elapsed since the teeth were removed and careful differentiation from the normal variations must be made.

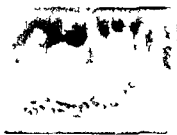
The following account of the various appearances which are recorded is

given with the full knowledge that the future may provide an explanation other than that put forward here

When a tooth is removed the socket becomes filled in with new bone and the lamina dura is absorbed with the result that after a varying period of time there is no evidence to show where the socket has been. There are cases in which many years after a tooth has been removed the radiogram reveals what appears to be a socket from a recent extraction—that is there is no change in the thickness and definition of the lamina dura and that there is no new bone in the cavity of the socket. Such sockets have been encountered sixteen years



FIG 601—Persistent sockets sixteen years after removal of the teeth



FIGS 602 603—Residual infection. Sockets with small amount of new bone formation



FIG 604—Residual infection. Sockets with new bone resembling retained roots



FIG 605—Residual infection. Rarefying cysts in the nasor region



FIG 606—Residual infection. Marked rarefaction of the alveolar bone

after extraction and investigation has shown the presence of pathogenic organisms (Fig 601). From this extreme there are variations in the degree with which the outline of the lamina dura persists associated with more or less new bone formation in the socket so that there may be revealed traces only of the socket cortex enclosing a rough caricature of normal bone structure (Figs 602 603). In other types the sockets may be completely obliterated and new bone of a very rarefied type present throughout the affected areas (Figs 605-606) or the only evidence may be a markedly irregular alveolar surface with or without rarefaction of the underlying bone. Any combination of the foregoing appearances may be met with and lead to suspicion of the



presence of residual infection. There is another type in which the new bone laid down in the sockets is found to be very hard leading to the erroneous view that roots are present. The appearance in such a case is that of persistent sockets with fairly well marked lamina dura inside which is a layer of bone not completely filling the socket but leaving a space in the centre closely simulating the appearance of the pulp canal in a root (Fig 604). This causes confusion but a tooth root is denser and more homogeneous than the new bone and the central space for the canal much narrower than that seen in the other condition.

This type offers difficulties in treatment because of the extreme hardness of bone in the sockets. The conditions just described may occur in all parts of the tooth bearing area at the same time or may be localised to one or more areas of which the lower incisor region is the most common. The significance of the condition may be local as a cause of pain under a denture or occasionally of chronic neuralgia. Its general significance is found in those cases in which the affected area is acting as a focus of infection. In attempting to recognise the appearances it must be remembered that the healing of a socket may be delayed if there has been much instrumentation or medication and consequently a liberal margin of time must be allowed to account for this. If any of the above appearances are present a year after tooth extraction the condition is considered to be due to residual sepsis.

A similar conclusion is formed if radiographic examinations made some time apart reveal no improvement in the appearances of the bone and sockets even though teeth may have been removed considerably less than a year and if this is supported by the presence of certain clinical appearances in the gums. At all times the normal variations must be kept in mind in order not to confuse them with the above conditions.

## DENTAL CARIES

Dental caries is usually recognised by visual and instrumental methods but there are many occasions on which even large cavities are not discovered except on radiographic examination. Radiography is used to reveal the presence of small interstitial cavities which are too small to be found by other methods but it is not uncommon for unsuspected extensive cavities to be found in the course of routine radiographic examination.

In cases of obscure neuralgia the greatest help is sometimes afforded by radiographic examinations in revealing among other causes hidden caries. There are special dental films termed Bite Wing films which are very helpful in obtaining radiograms of the crowns of the teeth.

The presence of a carious cavity is recognised by decalcification in the enamel and dentine revealed in a radiogram as a dark shadow (Figs 607-609). Care must be exercised in giving opinions as to the involvement or otherwise of the pulp chamber as the two shadows may only be superimposed by projection. The presence of anatomical depressions in the sides of the teeth often causes



FIGS 607-609 —Dental caries

FIG 610 —Pulp stone  
in the upper incisor

FIGS 611-612 —Productive perodontitis

FIG 613 —Rarefying  
perodontitis of  
the second premolarFIG 614 —Incompletely formed  
root of the  
upper incisorFIG 615 —Absorption  
of the distal aspect  
of the root  
of the incisor  
simulating  
dental cariesFIG 616 —Granuloma  
of the pulp

appearances which resemble those of decalcification — this is especially noticeable in the premolar and canine teeth. It may be impossible to differentiate between them from the radiograms alone.

Carious cavities which occur below the gum margin are also revealed in radiograms in many cases where their presence is quite unsuspected and a root

may be completely separated from the crown without any clinical manifestation. This applies also to caries beneath fillings. The appearances are similar in all cases. It must be pointed out that cavities occurring in the buccal or lingual aspects of teeth may not be revealed in a radiogram.

**Changes in the Pulp**—The size of the pulp chambers in the teeth of the young subject is larger than in those of older people, and this is due to the fact that there takes place a deposition of secondary dentine on the walls of the pulp chamber. This is not a pathological condition but can be regarded as a senile one. Degenerative changes may occur in the pulp and result in the deposition of lime salts forming small bodies which are termed "pulp nodules" or pulp stones. These appear as rounded or cylindrical opacities situated in the pulp chamber or root canal and are more commonly found in elderly people (Fig. 610). In teeth which have been subjected to injury the pulp chamber or root canal may become completely obliterated by the deposition of lime salts. This is a rare occurrence and is most commonly found in anterior teeth.

**Filling Materials**—All filling materials are opaque to the X rays, the metallic being more so than gutta percha and silicate ones. Almost all the materials used as fillings contain metallic substances and it is for this reason that they cast a shadow. Gutta percha points used in filling root canals contain metallic salts and are opaque but the pure gutta percha which is sometimes used in the treatment of fractures is not at all radio opaque.

**Foreign Bodies**—The only foreign bodies which are at all commonly found in the jaws are pieces of filling material root fillings and fragments of hypodermic needles.

If it is intended to attempt the removal of a broken hypodermic needle, careful radiographic localisation must be carried out as near the commencement of the operation as possible on account of the readiness with which some broken needles move in the soft tissues.

## DEAD TEETH

A tooth may be dead and reveal no evidence of its death in a radiogram, but frequently there are present appearances which definitely indicate or suggest with more or less certainty that the tooth is not vital.

The presence of filling in the root canal or pulp chamber is definite evidence that the tooth is dead. *Rarefying osteitis at the apex* is very strong evidence of death in the very great majority of cases for it is only very rarely that a tooth is found to be vital and yet reveal an area of bone destruction at its apex. This does not apply with the same force to those teeth which are involved in a dental cyst, for it is not uncommon for many such teeth to be found vital. The presence of a large carious cavity which extends to the pulp chamber is also strong evidence that the tooth is dead but it must be remembered that the two shadows may only be superimposed by projection, and not be in actual

communication. A less certain but distinctly suggestive appearance is thickening of the periodontal membrane localised to the apex of a tooth, an appearance which is found at the apex of some infected teeth, usually dead, but sometimes live ones.

### PRODUCTIVE PERIODONTITIS

(*Syn* exostosis and hypercementosis)

Productive periodontitis may occur alone or in association with apical osteitis. The condition is one of thickening of the root or roots of teeth and is probably due to irritation, infective or otherwise.

It has been suggested that in view of the fact that the teeth most commonly affected are the premolars, there may be some degree of occlusal trauma acting as an aetiological factor. It is a striking fact that the upper and lower premolars are very commonly affected in comparatively young subjects in whom there is no evidence of oral sepsis and who may not show similar changes in other teeth. Some observers believe the condition to be a chronic infective one.

It is rare to find the lower incisors or the upper central incisors affected and this fact suggests that occlusal trauma is not an important factor.

It is probable that chronic sepsis does play a causative part in some cases and the writer takes the view that teeth which are involved with productive periodontitis in a subject suffering from focal sepsis must be regarded as possible foci, but in healthy subjects such teeth may not be harmful. Both living and dead teeth are involved. Radiographically the condition is recognised by a thickening of the root, such thickening being fusiform, spherical or of irregular shape (Figs 611-612).

The new tissue may be indistinguishable from the parent tooth, but quite often it appears to have a diminished density as compared with the tooth root. The deposition of new tissue takes place entirely beneath the periodontal membrane, so that this membrane and the lamina dura are clearly seen. This differentiates the condition from sclerosing osteitis, which causes a condensation outside the periodontal membrane and which obscures it and the lamina dura. Productive periodontitis may be present but not revealed in a radiogram when the deposition has occurred on the buccal or lingual aspect of the affected tooth. In some radiograms of the molar teeth, especially the lower ones, there are encountered appearances which simulate productive periodontitis but which are due either to rotation of the tooth or to incorrect positioning of the X-ray tube, so that an oblique view is obtained. The fact that the view obtained is an incorrect one indicates that care must be taken in interpretation and re-examination may be necessary.

Many teeth when removed reveal a translucent appearance of the roots and this is said to be due to increased calcification. In a radiogram of such a tooth one might expect to see root shadows of increased density, but no such

change is demonstrated and the condition cannot be recognised. In some cases of productive periodontitis the new tissue which is laid down may extend from one tooth to involve another and result in false gemination.

### RAREFYING PERIODONTITIS

(*Syn* absorption of the root)

It is probable that under this heading several different processes are included. The condition is manifested by the absorption of the tooth root with or without evidence of other abnormality such as apical osteitis.

Absorption of the root of the deciduous tooth does not appear to come within this designation but when the tooth is a permanent one and the root is being absorbed by pressure from another tooth, possibly an unerupted one then the term does apply. A tooth with apical osteitis is frequently seen to have absorbed apices and the two conditions are ascribed to the same cause namely sepsis.

In another group of cases the apices of one or more teeth perhaps the whole dentition may be absorbed with no evidence of other abnormality and the teeth may be alive and apparently healthy. In some cases the apices are partially destroyed and in others completely the tooth sockets being filled with bone and lined with a new complete lamina dura. In other cases the socket whence the tooth has been absorbed remains patent. Patients have been examined clinically and found to have normal healthy mouths and teeth, but on radiographic examination have shown serious absorption of many of the teeth. Many such cases have been carefully investigated and pyogenic organisms have been found at the apices of the affected teeth.

In the present state of knowledge the condition must be regarded as a probable source of focal sepsis. It is manifested in a radiogram as a shortening of the root associated with a wide apical foramen (Fig 613) a point which is used to differentiate the condition from a foreshortening shadow due to faulty technique. In the latter case the tooth is short but the apical foramen is not wide. The presence of a tooth adjacent which is of normal length serves to emphasise the condition of true root absorption. A tooth affected by rarefying periodontitis is differentiated from one which has not finished its growth by the fact that in the former the root canal walls converge towards the apex whereas in the latter the converse is observed (Fig 614). Sometimes a radiogram of a dead tooth shows the root filling to be bereft of root but surrounded by apparently normal bone as if the root had never been present and the root filling placed in the crown and projected into the alveolus. This is another manifestation of rarefying periodontitis.

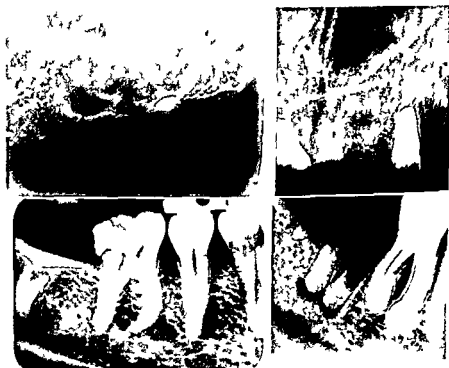
**Granuloma of the Pulp** (*Syn* pink spot)—At this place it is convenient to discuss another type of root absorption one that commences inside the tooth and is not due to periodontal disease.

The condition is sometimes termed *Pink Spot* and is due to a formation of granulation tissue in the pulp. As the result of this new tissue formation the adjacent portion of the tooth is absorbed usually producing a central circular defect in the root or crown (Fig 616). The condition has to be differentiated from perforation of the root as a result of faulty instrumentation and this is usually rendered possible by the absence of any history of instrumentation or by the fact that the lesion is too inaccessible to have been produced artificially. The condition is not only of academic interest for it may be so extensive as almost to separate the crown from the roots.

### BURIED ROOTS

Buried roots are often present quite unsuspected and may be associated with sepsis so that in cases referred for investigation of focal sepsis the edentulous areas must also be examined. The appearance of roots which have been retained in the alveolus does not vary from that of the whole tooth but there are occasions on which on account of their small size they may be difficult to recognise (Figs 617-620).

A small fragment of tooth root unassociated with bone sclerosis is usually easily recognised on account of the fact that some part of it is covered by perio-



FIGS 617-620 — Retained roots

dental membrane showing as a thin dark space separating the root from the lamina dura. Even very small spicules of root present an opacity which is homogeneous and are seen to be devoid of any structure resembling the surrounding bone. Their density is such that differentiation from sequestra can usually be made.

It is a rare occurrence for a root to be present and not to be shown in a radiogram but it does sometimes happen. In the presence of sclerosing osteitis it may be impossible to differentiate the presence of a root in the centre but it is sometimes possible to do so on account of the presence of the dark spaces which represent the root canal and the periodontal membrane. Careful study of roots often enables a diagnosis of the presence of a sharp typical hook to be made. If the hook is forwards or backwards it will be more easily recognised than in those cases in which it is directed inwards or outwards. The latter condition is sometimes revealed by the presence of a small circular dark line with a central dark spot situated over the end of a root and representing a periodontal membrane and the root canal of that portion of the root which is bent so as to lie in the plane of the incident beam of rays. Close scrutiny is necessary to recognise the condition.

The roots of third molars are sometimes abnormally placed and increased in numbers and thus may or may not be recognisable in radiograms. It is frequently impossible to recognise these small accessory roots even when they are known to be present. The type of absorption which resembles caries radiographically and which is encountered in some unerupted teeth may be present in buried roots in such a degree as to render the differentiation of the tooth structure from the bone very difficult.

It is helpful to a dental surgeon to be warned that such a condition is present before he commences operation.

### TARTAR

Deposits of tartar can be recognised either above the level of the gum margin or below, and such deposits may be large or small. Radiographic evidence is only sought in those cases in which there are small deposits of serumnal calculus hidden beneath the gum.

## CHAPTER LIV

### INJURIES TO THE TEETH AND JAWS

#### FRACTURES OF THE MANDIBLE

EXAMINATION OF any case of injury or suspected injury to the jaws is incomplete unless accompanied by careful radiographic study. Apart from the surgical point of view, the necessity to carry out an X ray examination is very important in order to avoid any legal imputation of neglect. The examination must be a complete one and include lateral views of both sides and a postero anterior one, including the condylar processes.

It is a rule in radiography that bone injuries must be examined in two planes, and the mandible is not an exception. For this reason three views must be obtained in order to ascertain the presence of an injury and to estimate the



FIG. 621 —Fractures of the left ascending ramus and in the right incisor region



FIGS. 622-623 —Fractures of the mandible



degree of deformity In the incisor region some difficulty is experienced in obtaining a clear uninterrupted view in an extra-oral radiogram, consequently a large intra-oral one is placed in the occlusal plane, in which excellent detail of the whole of the anterior portion of the mandible may be obtained With the complete set of radiograms study is made to ascertain the following points

**The Number and Sites of the Fractures**—One fracture only may be present but it is rather more common to find two or more (55 per cent according to Ivy)

The commonest sites are at the angles of the mandible, the canine region, and the neck of the condyle

It is the last region which is most commonly overlooked partly because it is not suspected and also because of the difficulty in recognising injury when it is present in that region If the angle or canine region is involved on one side it is common to find evidence of fracture at the neck of the condyle or in the ramus of the other side In cases of severe injury there may be three or more fractures present

**The Type of Fracture Present**—Nearly all fractures of the mandible are complete, but sometimes cases are encountered in which the radiographic appearance is that of an incomplete one The line of fracture may be vertical or oblique, and it is common to see an appearance suggesting that there are two lines of cleavage closely related, but this is due to the projection of the two edges of an oblique fracture, the broken inner and outer cortex each simulating a separate injury

Difficulty is sometimes experienced in recognising the presence of a fracture which has an oblique direction in the sagittal plane unassociated with displacement of the fragments Each fragment is wedge shaped and the rays do not pass through the line of cleavage except in the postero anterior view, and it is only in this position that such an injury may be recognised In presence of displacement of one fragment upon the other the recognition of the injury is rendered easy Comminuted fractures were common during the Great War, but they form a small percentage of civil cases In some cases the injury involves the alveolus only, and then it may be difficult to detect

Teeth may be injured with or without bony involvement, and it is of importance that this should be recognised especially in the former event and if the fractured root is related to the site of injury of the jaw

**The Amount of Displacement**—The displacement of the fragments may take place upwards, downwards, inwards or outwards depending upon several factors, and it is of importance to the surgeon to be able to see the exact state of affairs before any treatment is undertaken The direction of the fracture of the angle is important because it may play a part in the retention of the posterior fragment in the correct position In fractures of the condylar region it is the neck or base of the neck which is usually involved, and dislocation of the con-

dyle may occur in which case it may be displaced forward and be situated vertically in front of the eminentia articularis or may be turned on its side and displaced forwards and inwards or very rarely outwards

In direct injury upon the point of the chin both condyles are sometimes

FIG. 624 —Fracture of the mandible



FIG. 625 —Fracture of the alveolar border of the maxilla



FIG. 626 —Fractures of the base of the neck of the mandibular condyle



FIG. 627 —Extra-oral radiogram of a fracture of the mandible

involved. Intracapsular fractures of the condyle rarely occur. Fractures of the coronoid process are uncommon and are recognised by the displacement upwards of the process. If the tip only is involved it may not be displaced.

The Relationship of the Teeth to the Line of Fracture must be noted. The line of fracture frequently passes through the socket of a tooth and may

separate a root from the crown of a tooth. This is seen more commonly in the molar region. Some experts attach considerable importance to the removal of teeth which are very close to or in actual relationship with the line of fracture. Radiographic evidence suggests that, in those cases in which roots or teeth have been allowed to remain in the line of fracture infection and delayed union are more commonly encountered than in those where such roots have been promptly removed.

The presence of teeth in occlusion and situated upon the posterior fragment is also noted on account of its value in preventing displacement. The presence of unerupted teeth in the line of fracture is also of significance on account of a tendency to delay union.

The Presence of any Gross Abnormality, such as foreign body or bone disease which may or may not have contributed to the injury, is revealed and due consideration must be given to it. Pathological fractures of the mandible are usually the result of osteomyelitis or epithelioma or of trauma occurring whilst the treatment of these conditions is being carried out. Cysts of the jaw very rarely contribute towards the production of fractures of the mandible. Some cases of injury are followed by claims for compensation and the presence of any pathological condition which might have predisposed to the fracture must be recognised and its importance carefully estimated.

**The Progress of Treatment**—It is an interesting fact that in common with other head injuries union may occur and not be revealed in a radiogram, consequently no reliable opinion can be given as to the presence of union for some time after it is known to have occurred clinically.

Notwithstanding this much information can be gained by radiographic study during the course of treatment. Union is likely to be delayed if sepsis is present and this is frequently revealed by the irregularity and rarefaction of the edges of the fragments with or without sequestra in the line of fracture. Union is not likely to occur so long as there is a sequestrum between the fragments. In the presence of comminution care must be taken not to regard the small fragments as sequestra because removal of these islands of bone sometimes results in non union. Non union is revealed in a radiogram by the edges of the fragments becoming rounded off and losing their irregular margins and when these appearances are seen union does not take place without surgical intervention of some kind.

There is only one other appearance to be noted in the differential diagnosis of fractures of the mandible. Air present between the tongue and the soft palate and posterior pharyngeal wall is shown in a radiogram as a dark linear shadow, and this is sometimes mistaken for a fracture. The similarity may be very close, and the correct interpretation can only be ascertained when further radiograms are taken after the patient has swallowed or by the recognition that there is no breach in the continuity of the bone and that the dark line extends beyond the width of the bone.

IN CHILDREN certain factors arise which do not occur in adults

The presence of unerupted teeth in their crypts renders it much more difficult to recognise a line of fracture which is unassociated with displacement of the fragments, and it is sometimes impossible to see the line of cleavage. The presence of unerupted teeth in their follicles situated in the line of fracture does not appear to interfere with the progress of union, but on the other hand very serious consequences have followed the removal of such teeth from their crypts. Fractures of the condyle in children have an added importance because they are likely to be followed by ankylosis. *Ivy* states that this is not so, but the writer has seen this complication on several occasions always in children.

### FRACTURES OF THE MAXILLA

Fractures of the maxilla vary in extent from a simple injury of the alveolus to complete separation of the maxillæ from the cranium. Fractured alveolus is commonly encountered following a blow on the front of the mouth (Fig 625) and may be associated with injury of the incisor teeth. The teeth are sometimes seen to be displaced from their sockets but still in the alveolus with or without evidence of a line of fracture situated above the roots or across them. In the third molar region the tuberosity of the maxilla is sometimes detached during the operation of extraction of that tooth. The antral cavity may be opened by such an accident. In cases of more serious injury both maxillæ may be separated from the skull with very little radiographic evidence, indeed it is sometimes found to be impossible to recognise the condition. The separation usually occurs high up, and the lines of cleavage are through the nasal portions of the maxillæ and posterior walls of the antra. Injuries of the maxilla are often associated with fracture of the malar bone which is sometimes seen to be depressed into the antral cavity, the lines of fracture in such a case being situated in the zygomatic arch, the inferior margin of the orbit, and the outer and posterior walls of the maxilla. Sometimes there is also separation of the suture at the external angular process. Special views must be taken in order to show these fractures to the best advantage. With the plate placed under the chin and with the head extended as far as possible the central ray is directed downwards and backwards over the root of the nose 20 degrees towards the feet. The radiogram taken in such a manner shows the malar bones, zygomatic arches, inferior borders of the orbits, nasal processes and the walls of the antra. These fractures are further considered in the section on injuries to the facial bones.

### FRACTURED TEETH

Fractures of the crowns of teeth are usually easily recognised clinically, but occasionally radiographic evidence first draws attention to the injury. In the case of a fractured crown radiograms are sometimes taken in order to attempt

to show whether the pulp chamber is involved. Injuries of the roots of the teeth occur with or without injury of the alveolus and in routine examinations old and quite unsuspected fractures are sometimes revealed (Figs 628-629).

In injuries to the body of the jaw the line of fracture sometimes passes through the root of the tooth, separating it from the crown. The commonest tooth to be injured is the upper central incisor. The line of fracture may be transverse and associated with little or no displacement or very considerable



FIGS 628-630 —Perforation of tooth roots



FIGS 631-63 —Fractured tooth roots

separation of the fragments may be present. The coronal portion of the root may be pushed backwards with or without obvious injury to the alveolus. The line of fracture is not always shown, especially when it is a longitudinal one occurring in the coronal plane. Union sometimes occurs between the fragments. When a tooth has been subjected to severe trauma an area of rarefaction usually appears in the bone at the apex and this may persist as an infective process or may subside after a variable short period of time.

In a case in which recent injury has occurred it is difficult to express an

opinion as to whether an apical area of bone rarefaction is traumatic or infective. Teeth which have been injured before the growth of the apices has been completed usually die and cease to lay down more dental tissue. Such a tooth is recognised by the fact that the apex is more widely open than the rest of the root canal. This differentiates the condition from an absorbed apex.

**Perforation of the Root**—Perforation of a tooth root by instrumentation may or may not be easily discovered in a dental radiogram. The condition is very obvious when a post of a crown or portion of a root filling passes beyond the end or through the side of a root (Figs 630-631).

If no opaque material passes through the perforation the false opening is suggested by the presence of an area of osteitis at the side of a tooth, the root canal of which is seen to extend to the side of a root (Fig 632). In the absence of osteitis the abnormal position of the root canal shadow would make further close study necessary, when the actual orifice might be recognised. The presence of osteitis at the side of a root which has been subjected to dental treatment is always very suggestive of a root perforation, even when unassociated with other evidence, and further radiograms should be taken after attempts have been made to leave diagnostic wires in the root canal proper and in the artificial passage. In this way a perforation will often become apparent which cannot be shown in any other way (Fig 630). Difficulty is experienced in recognising the perforation of a root, when it is situated buccally or palatally, on account of the fact that the material which had perforated the side of the root may still appear to be in its correct position in the canal, an effect produced by the projection of the shadows and one which would not offer difficulty could a profile view be obtained. The perforation is usually recognised by the fact that it causes osteitis at its site of penetration and consequently a small dark area is seen to be present through the root and perhaps extending to one or both sides of it, but such an appearance is sometimes not easy to recognise.

The above is in effect a restatement that osteitis occurring at the side or middle of a root usually indicates the presence of a perforation.

Perforation of the floor of the pulp chamber in a molar tooth is usually recognised by the actual orifice or on account of the presence of opaque material or osteitis at the root bifurcation. The crown of a tooth is sometimes perforated at its cervical margin, and this is usually recognised by the presence of opaque material. The condition known as granuloma of the pulp sometimes extends to erode the side of a tooth, and may simulate a perforation. The symmetrical appearance of the absorption suggests the true state of affairs. The apex of a tooth may appear to have been perforated by an instrument when it has actually died before completion of growth. Such a condition is usually clearly indicated by the fact that the apical end of the root canal is funnel shaped, an effect which could not be produced by instrumentation.



FIG 633 Large dental cyst



FIG 634—Occlusal view of mandible showing expansion of the alveolar plates



FIG 635—Dental cyst arising the ascending ramus



FIG 636.—Dental cyst



FIGS 637-638—Dental cysts



FIGS 639—Cyst of anterior palatine foramen

healthy. In one such case examined small dental cysts extended from the second molar on one side of the mandible right round to the second molar on the opposite side. All the teeth appeared to be healthy and radiographic examination carried out a year previously did not reveal any evidence of abnor-

malty except impacted third molars which were removed. There was no clinical evidence of the condition which on histological examination showed the typical structure of a dental cyst.

Very rarely a dental cyst occurs so superficially and produces so little bone destruction that its presence is not revealed radiographically.

When dental cysts occur in the maxilla they may cause the antral floor to be pushed upwards and in extreme cases the air sinus is so completely obliterated that the floor of the orbit becomes the roof of the cyst. It is exceedingly rare for a dental cyst to open into the antrum in the absence of instrumental interference, but it may occur.

There is an interesting type of dental cyst which occurs in the anterior palatine foramen expanding it in a symmetrical manner (Fig 639). It may be impossible to differentiate a small dental cyst occurring in the upper premolar and molar region in an edentulous patient from a normal antral shadow by radiographic evidence alone and it is not at all uncommon for such a condition to be operated upon without proper consideration of clinical evidence with the result that a normal air sinus is opened. A normal antral shadow frequently closely simulates that of a dental cyst but errors in interpretation are not likely to be made if due importance is given to the clinical conditions present, a statement which cannot be overstressed. Occasionally a dental cyst is found at operation to be filled with a thick material which does not give any radiographic evidence of its presence.

## DENTIGEROUS CYSTS

(*Syn follicular odontome*)

These cysts occur usually in the mandible but are seen in the maxilla. The appearance of a dentigerous cyst is very closely similar to that of a dental cyst and differs only in that the former contains the whole or some part of an unerupted tooth (Figs 640-644). The area of bone destruction shows no evidence of structure within it except the tooth and the margin is clearly defined but not always corticated. If we leave aside the question of aetiology the radiographic evidence suggests that there are two types of dentigerous cyst—one which arises from infection of the follicle of a permanent tooth through the agency of an infected deciduous predecessor and another type which occurs behind the alveolus containing the deciduous teeth i.e. in the molar regions.

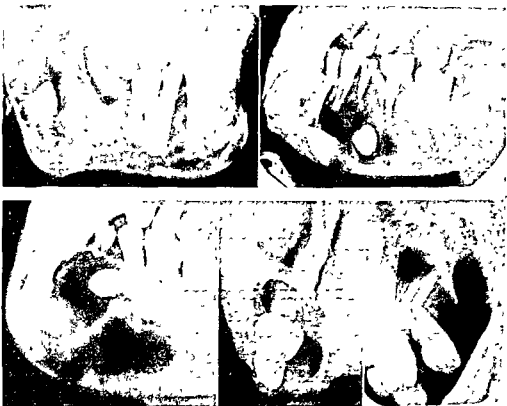
Dentigerous Cysts occurring in the Molar Regions usually have a white cortical margin whereas those in the other area usually have not. The point is of interest but probably has no practical significance.

**Infected Follicle**—As in the case of dental cysts the stage at which an infected process in the follicle of an unerupted tooth is designated a dentigerous cyst is quite arbitrary.



The follicle of an unerupted permanent tooth sometimes becomes infected by the deciduous predecessor and one of three things appears to occur.

*In the case of acute infection suppuration may take place and the whole of the follicle become involved, causing destruction of the cortical lining with death of the tooth, which becomes a sequestrum. This condition is revealed radiographically by destruction of the white cortical line, by the rarefaction*



FIGS. 640-644.—Dentigerous cysts.

of the bone adjacent to it, and usually by displacement of the partially formed tooth.

*In those cases in which the infection is less acute the involvement of the follicle may stop short of the growing end of the tooth and the condition subside without damage to the permanent tooth, which eventually erupts normally.*

The radiographic appearance of such a condition is shown by destruction of the cortical layer of some part of the follicle, with little if any rarefaction of the adjacent bone. In those cases in which the bone destruction extends to the growing end of the tooth no further growth will take place, eruption occurs, and the tooth becomes exfoliated.

The third possibility is that the infection will cause destruction of the wall of the follicle in a progressive manner so that the bony cavity becomes considerably enlarged and forms what is in fact a dentigerous cyst. Occasionally the last condition is accompanied by periostitis at the lower border of the mandible beneath the cystic cavity and this clearly indicates that the condition is an infective one.

Radiographically this latter complication is revealed by the presence of a layer of new bone formation occurring at the inferior border of the mandible.

Any temporary tooth which has apical osteitis may cause some slight rarefaction of the adjacent cortex of the follicle of the permanent successor but usually this does not result in obvious infection of the bony cavity.

### COMPOUND COMPOSITE ODONTOME

These are nearly always found in children or young subjects. There are cystic and non-cystic types the latter being the more common.

The Cystic Type presents an appearance which very closely resembles that of a dental cyst and only differs from it in that it contains varying numbers of small denticles or small fragments of tooth substance. The area of bone destruction is clearly defined and is demarcated from the surrounding bone by a definite cortex. The number of denticles varies from a few to a large number and in shape and appearance there is a wide variety ranging from little tooth-like forms to small shapeless fragments of dental tissue.

The Non-cystic Type is the commoner of the two and is found in any part of the jaws. Such a tumour is seen to be made up of a group of irregular opacities obviously consisting of dental tissues and more or less closely set (Figs 645-647). There is usually a small dark space surrounding the tumour as a whole or each individual portion of the tumour indicating the presence of a capsule. The size of the tumour is very variable. The cystic type is differentiated from the dental or dentigerous cyst by the presence of the small denticles or cleft dental bodies. The non-cystic type is differentiated from a complex composite odontome by the fact that it is made up of more than one fragment of dental tissue.

### COMPLEX COMPOSITE ODONTOME

This tumour varies considerably in size small ones being found fairly commonly usually in those regions where supernumerary teeth occur namely, in the upper incisor region (Fig 649). Large complex composite odontomes are rare. They are made up of masses of irregularly arranged tooth substances dentine cementum and enamel and on account of their dense structure they cast a shadow resembling that of a tooth except in form and denser than that of bone. The appearance of an irregularly formed mass of very dense substance sharply differentiated from the surrounding bone is the typical appear-

ance of a complex composite odontome and there is little with which it can be confused. It is not possible to differentiate the various substances which go to make up the tumour. The large complex composite odontomes present a small dark space which separates the tumour from the bone and indicates the presence of a soft tissue capsule (Fig. 648). This structure cannot be recognised in



FIGS. 645-647.—Compound composite odontomes.



FIGS. 648-649.—Complex composite odontomes.

radiograms of the small complex odontomes, but when present it is of value in establishing the differential diagnosis from an ivory exostosis, although this has not the density of an odontome.

### GEMINATED ODONTOME

Geminated odontomes are usually recognised in a radiogram, but it is not always possible to state definitely whether the teeth are fused or not.

It may be possible to offer a definite opinion if radiograms are obtained in different planes and from varying angles (Fig 650)

### DILATED ODONTOME

These odontomes are teeth which in their process of development have become distorted by dilatation of the whole or some part of the pulp canal (Figs 651-652)



FIG 650 — Geminated odontome



FIGS 651-652 Dilated odontomes



FIGS 653-654 — Enamel nodules

FIG 655 — Osteoma

The condition is easily recognised radiographically. There is usually a fairly normal coronal portion but the root has a central dilatation and assumes a hollow spherical shape wide open at its apical end.

### ENAMEL NODULE

An enamel nodule is the name given to a small cusp like mass of dentine covered with enamel which usually occurs at or near the junction of the roots of upper molars but is sometimes found on single root teeth.

The radiographic appearance is that of a small well-defined area of increased density if the shadow of the nodule is projected through the crown and as a small protuberance from the tooth when it is projected in profile. No other radiographic appearance resembles it (Figs 653-654)



FIGS 656-657—Epithelial odontome adamantoma



FIG 658 Epithelial odontome adamantinoma



FIG 659—Epithelial odontome multilocular type



FIG 660—Epithelial odontome multilocular type



FIG 661—Epithelial odontome solid type

## EPITHELIAL ODONTOME

(*Syn* multilocular cyst)

This condition occurs most commonly in the mandible usually in the posterior part of the body and in the ascending ramus frequently extending to and destroying the coronoid process

There is no one appearance which is typical of the condition but there are a variety of different ones and any combination of these may be present at the same time *The commonest appearance* is probably that of a large area of bone destruction situated at the angle of the mandible and involving the greater part of the upper border of the body and the anterior part of the ascending ramus. The line of demarcation of the bone destruction is sharp and the diagnosis is made on the presence of a few coarse and irregular strands of bone in the cavity. It is the coarseness of these trabeculae of bone which suggests the diagnosis (Figs 656-658). There is in this type no radiographic evidence of any loculation.

Associated with this or arising independently *there is another type* of radiographic appearance namely a localised honeycombed area of bone destruction. This zone is quite well defined from the adjacent bone and the individual bone spaces may not be at all symmetrical in size (Fig 661). In one case which had a very long history the cellular formation was very marked and each individual space was very nearly the same small size.

*A third type* is one which can really be described as multilocular in appearance i.e. there are well-defined cavities present separated by thin but fully formed septa. This type may be found to contain a buried tooth (Figs 659-660). As previously stated a combination of the three types may be found associated and it must be understood that the term *type* is only used to describe different appearances of the same pathological condition.

The writer has encountered one case in which malignant changes were clearly demonstrated in one wall of a large multilocular cyst. There is an interesting feature concerning these cysts namely that often the actual size of the cyst suggests a greater degree of bone destruction than is revealed radiographically.

## OSTEOCLASTOMA

(*Syn* benign giant celled tumour)

There are several different appearances presented by these tumours. No one description will cover all osteoclastomata. *One type* of radiographic appearance resembles a dental cyst in so far as there is a dark central cavity but the line of demarcation is not so clearly defined and may be replaced by several incomplete cortical lines suggesting that the bone had made more than one attempt to limit the tumour. There is no evidence of infiltration. The central cavity is not devoid of structure as in a dental cyst for there are a few fine

irregular trabeculae present which may need careful scrutiny for recognition. The diagnosis is established on the presence of these trabeculae and the indefinite cortex (Figs 663 and 665)

Another type reveals itself as a collection of small closely set cavities in the bone, with clearly defined walls but without any condensation to suggest the presence of a cortical lining. The individual cavities do not reveal any evidence of structure (Fig 662)

FIG 662

FIG 663

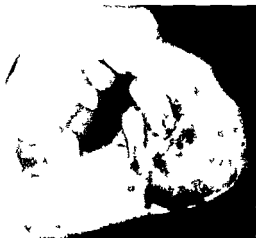


FIG 664

FIG 665

FIG 666



FIGS 662-666 Osteoclastomata

A third type resembles the two previous ones in that there is a tendency towards the formation of a large cavity with trabeculae which are coarser than those described above and unassociated with any cortication (Figs 664 and 666)

Associated with the large cavity are smaller ones similar to those found in the second type. These appearances may be found modified and the types described must only be regarded as attempts to indicate how the same tumour may appear in different individuals. Some osteoclastomata cannot be differ

entiated from other conditions on radiographic evidence alone. All osteoclastoma tend to cause expansion of the jaw i.e. the outer border will be seen to be covered with bone which is usually thicker and less smooth than that of a dental cyst. There is also a tendency for these tumours to push developing teeth away.

The differential diagnosis from a dental cyst is made from the absence of any structure in the dental cyst, its corticated margin and the smoothness of the expanded outer wall. A multilocular cyst has coarser and usually fewer trabeculae. It may be impossible to differentiate a fibrosarcoma but any evidence of infiltration suggests that the tumour is a malignant one.

## MYELOMATOSIS

(*Syn myeloma*)

This condition is sometimes encountered in the jaws and may be seen there before it is recognised elsewhere. Clear cut areas of bone destruction with no internal structure or cortical lining situated some distance from the tooth apex or at the apex of what appears to be a live and healthy tooth should evoke suspicion and lead to further investigations.

## CARCINOMA

Both carcinoma and sarcoma occur in the jaws the former being more common than the latter. *Epithelioma* may occur anywhere but the commonest site is at the junction of the anterior margin of the ascending ramus with the body of the mandible. The appearance which it presents is that of an area of bone destruction with no structure in the cavity and with no evidence of reaction on the part of the surrounding bone. The edges of the bone of the affected area are irregular indicating the presence of infiltration (Figs 667-670).

There is a *second type* of carcinoma which is occasionally encountered in the jaws and which histologically reveals the appearance of basal celled carcinoma. In the early stages the appearance is indistinguishable from that of an unhealed tooth socket except that there is no evidence of lamina dura or of any new bone formation (Fig 671). Suspicion should be raised by such appearances and in the absence of a satisfactory history or of change after a period of time histological section may be necessary to establish the diagnosis. If left untreated wide destruction of the jaw may result (Fig 672).

*Secondary carcinoma* appears to be a rare disease in the jaws and presents an appearance which is sometimes mistaken for osteomyelitis but the true nature of the condition is suggested by a more or less localised area of central destruction with an indefinite margin and some bone structure in the involved area yet with no reaction in the surrounding bone. The absence of sequestrum and the history differentiate the condition from chronic osteomyelitis.





FIG. 667.—Epithelioma of the alveolus with antral involvement.



FIG. 668.—Epithelioma of the mandible.



FIGS. 669-670.—Epithelioma of the mandible.



FIG. 671.—Basal-celled carcinoma of the mandible.



FIG. 672.—The same case, some months later.



FIG. 673.—Sarcoma of the maxilla.



FIGS. 674-675.—Two cases of sarcoma of the mandible.



FIG. 676.—Sarcoma of the mandible.

FIG. 677.—Same case as Fig. 676, two months later.

### SARCOMA

Sarcoma may be indistinguishable from epithelioma, or it may present quite a different appearance due to the presence of bone structure. This bone structure varies from a few trabeculae irregularly arranged in an area of bone destruction to a dense mass of calcification with no bone destruction (Figs 673-677)

*In the former*, malignancy is suspected by the fact that the tumour may show obvious evidence of infiltration of the adjacent bone or of invasion of the antral cavity, this contrasting with a cyst or osteoclastoma both of which cause the floor of the sinus to be displaced upwards.

*The osteogenic type* of tumour may in its early stages be almost indistinguishable from a simple osteoma, but the presence of any irregularity in outline or of bony spicules on the surface renders investigations advisable even without the aid of clinical factors and history (Figs 676-677)

### OSTEOMA

This is a rare condition in the jaws proper.

The sites at which osteoma does occur are the angle of the mandible and the inferior border of the orbit. The appearance is that of a dense bony mass pedunculated or sessile (Fig 655). The tumour is much denser than the adjacent bone and lacks the definite uniform structure. Its margins are clearly defined.

## CHAPTER LVI

### INFLAMMATORY AND OTHER DISEASES OF THE JAWS

#### OSTEITIS FIBROSA

THIS is a relatively rare condition in the mandible and maxilla. The cystic type of the disease does not appear to affect the jaws but there are two different appearances which do occur and which on histological examination reveal the structure of osteitis fibrosa. There is *one type* which is most commonly found in the maxilla and which radiographically shows much new bone formation with a tendency to encroach upon the antral cavity. The appearance is that of homogeneous bony structure simulating the stippling of the rind of an orange (Fig 678). The condition may be extensive and when investigated surgically the new bone is easily removed but tends to recur. *The other type* is that of a slow growing enlargement of the mandible or of the alveolar border of the maxilla. The newly formed bone lacks the structure of normal bone is much denser and is devoid of uniform trabecular arrangement (Figs 681-682). The appearance most likely to be present is a granular or closely set mottling with here and there areas of diminished density which do not however resemble cysts.

In the maxilla new bone continues to grow and may occlude with the mandible and so prevent normal closure of the jaws (Fig 683). On surgical interference the new bone is found to be very hard indeed.

Osteitis fibrosa associated with parathyroid tumours may cause cyst like cavities in the mandible but general rarefaction of the bone is not present.

#### ACROMEGALY

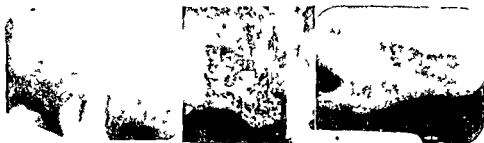
There is demonstrable change in the structure of the bone of the jaw in this condition.

Deficiency diseases do not produce any radiographic evidence of their presence.

#### OSTEOMYELITIS

Osteomyelitis of the jaws simulates the condition found in other bones in every important detail except two—the absence of chronic changes so commonly seen in long bones and of any marked degree of involucrum formation.

It must be remembered that acute osteomyelitis may be present and quite easily recognised clinically but no radiographic evidence be presented until



Figs 678-680—Osteitis fibrosa of the jaws



Figs 681-682—Osteitis fibrosa of the jaws



FIG 683—Osteitis fibrosa of the jaws

FIG 684—Leontias osea of the mandible

some time has elapsed on account of the fact that it takes a variable time (up to ten days) for sufficient bony absorption to occur to produce radiographic changes. Therefore to wait for radiographic evidence before establishing a diagnosis might lead to serious results. The earliest change in acute osteomyelitis may be a small area of osteitis at the apex of a tooth, followed by other areas of rarefaction occurring a little distance from the tooth and separated from it by what appears to be normal bone. Similar areas very quickly occur, extending along the bone until it may be involved in its entirety (Figs 687-688). In other cases the first evidence is a localised blurring of the bony trabeculae, quickly followed by small areas of bone destruction occurring separately over the affected area. The condition may be localised from the beginning, and this is usually the case of the maxilla, whereas in the mandible it sometimes spreads rapidly to involve a large area.

Different appearances are encountered in the subsequent stages, and any combination or modification of these may be found in any individual case.

*In the localised type* the whole of the diseased portion of the bone may be separated as a sequestrum from a central cavity, or the dead bone may be thrown off in small pieces over a period of time.

*In the more extensive cases* the diseased bone becomes broken up into small sequestra which may remain for a considerable time (Fig. 689). The appearance at this stage is a common one and is that of a jaw which is normal in shape but the texture of which is completely altered, so that there are multiple irregular cavities in the bone separated by fragments which may retain a close similarity with normal bone but which are obviously being separated. It is usual to see a small amount of periosteal new bone at the inferior border of the mandible only and it is significant that it is not seen elsewhere, and that it is not nearly so marked as in the case of a long bone. Indeed, in some cases there is no involucrum revealed at all. In the maxilla no evidence of involucrum is shown. At the stage in which the dead bone has been separated the appearance is usually that of the remains of the body of the jaw with no evidence of new bone formation except at the lower border of the mandible. Later on new bone formation at the site where the sequestra have been thrown off is revealed, and a radiogram at this stage would not present any features which are diagnostic of any one condition, and in the absence of history and clinical findings would be more or less meaningless.

When the disease is ended and the reparative process completed there is usually no evidence of any abnormality even in most extensive cases. Chronic changes as revealed by dense sclerosis in osteomyelitis of the long bones is not encountered in the jaws. Sometimes osteomyelitis is associated with and complicated by a pathological fracture, in which case there is added to the foregoing appearances evidence of the fracture with or without displacement of the fragments.

If the fracture has occurred early, displacement is likely to follow on ac-



Figs. 678-680 —Osteitis fibrosa of the jaws



Figs. 681-682 —Osteitis fibrosa of the jaws



FIG. 683 —Osteitis fibrosa of the jaws

FIG. 684 —Leontas ossea of the mandible

some time has elapsed on account of the fact that it takes a variable time (up to ten days) for sufficient bony absorption to occur to produce radiographic changes. Therefore to wait for radiographic evidence before establishing a diagnosis might lead to serious results. The earliest change in acute osteomyelitis may be a small area of osteitis at the apex of a tooth followed by other areas of rarefaction occurring a little distance from the tooth and separated from it by what appears to be normal bone. Similar areas very quickly occur extending along the bone until it may be involved in its entirety (Figs 687-688). In other cases the first evidence is a localised blurring of the bony trabeculae quickly followed by small areas of bone destruction occurring separately over the affected area. The condition may be localised from the beginning and this is usually the case of the maxilla whereas in the mandible it sometimes spreads rapidly to involve a large area.

Different appearances are encountered in the subsequent stages and any combination or modification of these may be found in any individual case.

*In the localised type* the whole of the diseased portion of the bone may be separated as a sequestrum from a central cavity or the dead bone may be thrown off in small pieces over a period of time.

*In the more extensive cases* the diseased bone becomes broken up into small sequestra which may remain for a considerable time (Fig. 689). The appearance at this stage is a common one and is that of a jaw which is normal in shape but the texture of which is completely altered so that there are multiple irregular cavities in the bone separated by fragments which may retain a close similarity with normal bone but which are obviously being separated. It is usual to see a small amount of periosteal new bone at the inferior border of the mandible only and it is significant that it is not seen elsewhere and that it is not nearly so marked as in the case of a long bone. Indeed in some cases there is no involucrum revealed at all. In the maxilla no evidence of involucrum is shown. At the stage in which the dead bone has been separated the appearance is usually that of the remains of the body of the jaw with no evidence of new bone formation except at the lower border of the mandible. Later on new bone formation at the site where the sequestra have been thrown off is revealed and a radiogram at this stage would not present any features which are diagnostic of any one condition and in the absence of history and clinical findings would be more or less meaningless.

When the disease is ended and the reparative process completed there is usually no evidence of any abnormality even in most extensive cases. Chronic changes as revealed by dense sclerosis in osteomyelitis of the long bones is not encountered in the jaws. Sometimes osteomyelitis is associated with and complicated by a pathological fracture in which case there is added to the foregoing appearances evidence of the fracture with or without displacement of the fragments.

If the fracture has occurred early displacement is likely to follow on as





FIGS 685-686 — Localised necrosis of the alveolus



FIGS 687-688 — Osteomyelitis of the mandible

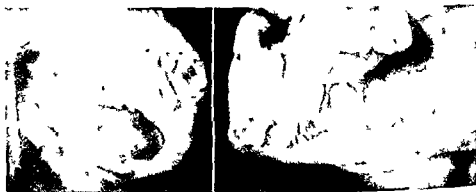


FIG 689 — Osteomyelitis of the mandible

FIG 690.—Periostitis of the mandible

count of the fact that no involucrum has been formed whereas later periosteal new bone may suffice to act as a splint to prevent displacement

The marked degree with which extensive osteomyelitis of the mandible is followed by complete resolution with absence of deformity is very striking

Syphilitic and Phosphorous necrosis may differ from the above description only in that there is a greater tendency for the condition to be localised and for the sequestra to be massive

### NECROSIS

Small localised areas of necrosis are frequently found in the jaws and the central sequestrum may be a part or whole of a tooth socket a fragment of root or dead bone (Figs 685-686)

It is rare to find necrosis produced by arsenical compounds used in devitalisation of the pulp but the condition is sometimes experienced The appearance in such a condition is usually that of a small localised area of bone destruction with a central rarefied sequestrum situated either at the apex of a root or at the surface of the alveolus adjacent to the cervical edge of the carious cavity

### PERIOSTITIS OF THE MANDIBLE

This condition usually occurs in children or young adults and the commonest cause is infection at the apices of one of the permanent teeth usually a molar The infection commencing at the apex extends outwards penetrates the outer plate of the alveolus and tracks beneath the periosteum The radiographic appearances are those of new bone formation at the inferior border of the mandible separated in the active conditions from the jaw by a thin dark line (Fig 690) This dark line is said to disappear when the disease becomes quiescent In some cases there is no evidence of any dental cause and the condition is considered to be an idiopathic periostitis

## CHAPTER LVII

### DISEASES OF THE TEMPORO MANDIBULAR JOINT AND ANTRUM

#### INJURIES TO THE MENISCUS "CLICKING JOINT"

PROBABLY THE commonest abnormality which results in the temporo mandibular joint being radiographed is that known as clicking jaw a name given to the condition in which movement of the mandible is accompanied by a clicking noise more or less loud and audible to the patient alone or to those in the presence of the patient. In the great majority of cases no abnormality is demonstrated in the radiograms of the joint but in a few a diminution in the width of the inter articular cartilage is shown by a narrowing of the distance between the head of the condyle and the surface of the glenoid fossa. It is probable that the change is the result of continued injury to the cartilaginous meniscus resulting from traumatic arthritis.

#### ARTHRITIS

Pain in the temporo mandibular joint associated with a clinical diagnosis of arthritis is not infrequent but the presence of radiographic changes is rare. Changes do occur which are described as being due to osteoarthritis or infective arthritis.

Osteo-arthritis presents diminution of the joint space usually without changes in the condyle itself but occasionally associated with flattening of articular surface. Osteophytic formation does not appear to occur or alternatively if it does it is not revealed in a radiogram.

There is no evidence of any rarefaction in the head of the condyle in osteoarthritis.

Infective Arthritis is recognised by the presence of definite rarefaction in the head of the condyle usually localised to its anterior aspect. The joint space is usually narrow. The condition is a very rare one.

#### ANKYLOSIS OF THE TEMPORO MANDIBULAR JOINT

Ankylosis of the temporo mandibular joint usually arises in children and young adults and results from injury or infection.

The injury may have occurred during birth. Ankylosis of the joint does not appear to follow injuries in the adult and its occurrence in children is probably due to the rapidity with which new bone is laid down with little stimulus during the period of active growth. Careful and complete radio-

graphic examination is necessary in order to ascertain the greatest amount of information and such an examination is rendered difficult on account of anatomical changes which impede clear access to the parts

The condition may be unilateral or bilateral. The radiogram reveals obliteration of the joint space marked flattening and enlargement of the head of the condyle which may be indistinguishable on account of its involvement in a mass of new bone. This new bone is firmly attached to the base of the skull and extends to envelop the neck of the condyle and a varying amount of the sigmoid notch.

It is sometimes found that the sigmoid notch and the base of the skull are continuous and the original site of the condyle is distinguishable only on account of the increased density of the bone.

In other cases the sigmoid notch appears to be accentuated by elongation of the coronoid process.

No evidence of fusion between the coronoid process and the maxilla may be demonstrable, although this is said to occur in some cases. Other changes occur in the mandible the affected side being shorter and less developed with teeth present high up on the ascending ramus.

The ascending ramus is shorter than normal and the notch in the inferior surface just in front of the angle is considerably deeper. The angle which the ascending ramus makes with the horizontal ramus is more acute than in a normal case. The chin is retracted and has an inclination from above downwards and backwards. In bilateral conditions the changes in the size and shape of the mandible are more marked.

## DISLOCATION OF THE TEMPORO-MANDIBULAR JOINT

This may be partial or complete unilateral or bilateral and with or without associated injury. It is most commonly found associated with a fracture of the neck of the condyle and is sometimes discovered quite unexpectedly in radiographic examination of other conditions.

In complete dislocation unassociated with injury the head of the condyle is shown to be situated in front of the eminentia articularis whereas in incomplete subluxation the head of the condyle rests upon the eminentia when the mouth is closed.

The condition is not difficult to recognise radiographically but the diagnosis of the incomplete dislocation is not conclusive unless examination is made with the jaw closed. In fracture-dislocation the condyle is either displaced forwards and remains vertical or is displaced horizontally inwards and forwards or more rarely outwards. It is not always easy to recognise the exact position of the head of the condyle and sometimes all that may be said is that a dislocation is present.

### HYPERTROPHY OF THE CONDYLE

This is a condition which is more frequently diagnosed clinically than found radiographically. It is a very rare condition and diagnosis is not accurate unless it is supported by definite radiographic evidence. The condition may be a local hyperplasia involving the condyle with enlargement of the glenoid fossa or it may be associated with increase in the size of the ramus of the affected side of the mandible.

Hypertrophy of the condyle causes protrusion of the mandible forwards. Hyperplasia occurs in the mandible without involving the condyle causing protrusion to take place so that the lower teeth bite outside the upper ones. In one case examined the condition arose in a young man and progressed slowly and very markedly so that when the jaw was closed the molar teeth in the two jaws were separated by a distance of three quarters of an inch and the lower incisor teeth occluded well outside and above the upper ones. All the changes appeared to be confined to the ascending ramus which were considerably length-

ened without any evidence of change in the texture of the bone. The teeth were not spaced indicating that no growth was occurring in the horizontal ramus. Radiograms of the pituitary fossa in this case did not reveal any abnormality.

Another case presented changes in the horizontal ramus only while another patient has been examined in whom the angles of the ramus have been too obtuse with the result that the anterior teeth do not meet.



FIG 65 Root in the maxillary antrum



FIG 69 One root in the antrum and one in the alveolus

### ANTRAL CONDITIONS

The radiographic examination of the air sinuses is dealt with elsewhere but there are some points concerning the antrum which are of special concern to the dental surgeon. These will be dealt with here.

On account of the frequency with which the roots of teeth are closely related to the antrum it is a common occurrence for the sinus to be opened during tooth extraction. In such a case the intra oral radiogram may reveal a breach in the cortical wall of the floor of the

antrum, a portion of which is absent, but in the case of a small opening through one of the sockets of a molar it may be impossible to demonstrate any defect in the antral floor. Sometimes the whole of the tuberosity of the maxilla is fractured, and may be removed and the antrum involved as a result. When the tuberosity has been removed the defect will be obvious, but it is more difficult to recognise a fracture without loss of bone substance.

A root of a tooth is frequently displaced into the antrum (Figs. 691-692), and more rarely the whole tooth may be forced into the cavity. A root is usually easy to recognise unless very small and obscured by the shadow of the malar bone. Intra-oral dental and large extra-oral sinus radiograms must be taken, and the position of the root accurately determined. The differential diagnosis is between a displaced root in the alveolus and one in the antrum, and it is established by the absence of a lamina dura around the tooth in the latter position (Fig. 692).

The antrum is involved in some cases of malignant disease arising in the alveolus, and the appearance is that of the tumour together with a breach in some part of the antral wall. Innocent tumours and dental cysts may invaginate the antrum, but actually open into it only in rare cases.

Osteomyelitis of the maxilla may spread to involve the antrum and result in a persistent opening between the mouth and the air sinus.

*PART FIVE*

THE EYE

BY

E. W. TWINING, M.R.C.P., F.F.R., D.M.R.E.,

AND

S. COCHRANE SHANKS, M.D., F.R.C.P., F.F.R.

## PART FIVE

### THE EYE

#### CHAPTER LVIII

##### THE EYE\*

THE CORNEA is visible in a lateral view only. The oblique projection of *Belot* eliminates most of the bony shadows which obscure the orbit in the sagittal view. In this the face rests on the film, the head turned 45 degrees towards the side in question. The central ray is normal to the film, and passes through the temporal region just behind the fronto-malar suture. Only a thin plate of bone is interposed between the tube and the eye in this view (Fig. 693). *Vogt's* method of introducing a small film into the conjunctival sac shows the anterior chamber clearly. The posterior part of the globe of the eye can be seen only if it is outlined by the injection of a contrast medium into Tenon's capsule, either lipiodol, air, or thorotrast.

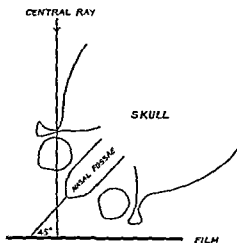


FIG. 693.—Belot's projection.

**Calcification of the Lens.**—This is a rare occurrence. In *Hartmann's* three cases it was in the form of an outer shell. It is clearly seen in a conjunctival "bone-free" radiogram or in *Belot's* projection.

**Ossification of the Vitreous.**—This also is rare. *Rollet* first described it in 1913. The degree of calcification is variable; sometimes a localised central shadow; sometimes a diffuse one extending to the periphery; sometimes a laminated shadow.

**Atheroma of the Carotid or Ophthalmic Artery.**—This may be seen, when calcified, as a ring shadow inside the orbital shadow, and in the case of the ophthalmic artery, in the optic foramen.

**A Shrunken Calcified Globe** may cast a dense intra-orbital shadow.

\* This part was begun by E. W. Twining, and, after his death, was completed by S. C. Shanks



## FOREIGN BODIES IN THE EYE OR ORBIT

Only the opaque ones can be shown radiographically. A doubtful speck on a film should always be checked by a second radiogram lest it be a film or screen fault. Opaque foreign bodies if not too small may be seen radioscopically and their movements related to the ocular movements.

**Types of Foreign Bodies**—Foreign bodies in the eye usually consist of small sharp particles of metal (most commonly steel) which are thrown off during hammering or other metal working operations. Some copper alloys and brass are brittle and from them may be thrown off extremely sharp particles with considerable force. Large industrial towns or shipyards produce the majority of examples. Most are superficially embedded in the cornea and are easily seen and removed. By far the commonest foreign bodies are tiny spicules of steel. These are nearly always clearly visible radiographically. Pearly, an extremely thin flake might be invisible if viewed face on.

*Jung* has pointed out that the bed of a foreign body which had been removed after being in the eye for some time may become impregnated with metallic oxides and cast a faint shadow. This siderosis if marked may also spread and outline the whole of the globe (*Genet*).

Chips of glass and spicules of cement or stone may cast a shadow. Spicules of lead glass certainly will. Even wood has been known to cast a slight shadow (*Toole*). There is more chance of this if the spicule has come from the surface of painted wood.

## LOCALISATION OF FOREIGN BODIES IN AND AROUND THE EYE

Serious responsibility rests upon the radiologist called upon to examine a case in which there is a history of damage to the eye with or without evidence of an inflamed eye. The great majority of intra ocular foreign bodies are of pin head size or smaller. These are the most important to localise and remove. The larger ones are apt to damage the eye irreparably so that it cannot be saved. Penetration may be through the cornea into the anterior chamber or through the iris also and thence into the posterior chamber or directly into the posterior chamber via the sclerotic.

Two steps must be taken in examining these cases

- 1 Detection of the shadow of the foreign body
- 2 Its localisation

**Detection**—This is done by means of simple films without a grid. Screens are used. The patient should look straight before him motionless. Two lateral films should be made using different screens for each film with the object of excluding artefacts. A short exposure is essential. Postero anterior films are usually of little value in detecting minute foreign bodies but should be taken as a routine since the size of the foreign body is at this stage unknown.

If a large one is shown clearly outside the orbit, this finding in combination with the clinical signs and history may remove all uncertainty

**Localisation**—It is of first importance to be able to orientate the foreign body, not to the skeleton but to the globe of the eye itself, particularly the tiny ones. The larger ones if intra ocular will have destroyed the function of the eye. Again the localisation must be precise to about a millimetre to minimise the amount of surgical trauma when the foreign body is removed

For satisfactory localisation the following conditions must be fulfilled

(1) THE FOREIGN BODY MUST BE VISIBLE

(2) THE HEAD MUST BE IMMOBILISED

(3) THE EYE MUST BE IMMOBILISED relatively to the orbit either by closing the eyes and assuming the position of sleep or preferably by fixing the eye on a distant point—either the injured eye or the sound one if the vision of the injured eye has been destroyed

(4) THE EYEBALL OR SOME POINTS ON IT MUST BE RENDERED VISIBLE  
Many methods have been used for this

(a) Conjunctival film for anterior chamber

(b) Injection of lipiodol thorotrast or air into Tenon's space

(c) Certain points on or close to the eye may be marked and the outline of the rest of the globe deduced from the assumption that its average diameter is 24 mm. Allowance must be made for cases of marked ametropia or collapse of the eye from the wound

Numerous localising markers have been used such as a lead ball held 1 cm from the cornea, a blepharostat carrying a metal ring, hooks or neurosurgical silver clips attached to the conjunctiva, a silver wire circle introduced into the conjunctival fornices and bearing cross wires in front of the cornea, tiny lead pellets fixed to the limbus, a small spring ring placed on the conjunctiva at the insertion of the ocular muscles, wire contours of the eyeball held over it during radiography in the postero-anterior and lateral views, contact glasses bearing an opaque line marking the limbus or cross lines over the cornea or opaque dots spaced at 90 degrees round the limbus

The centre of rotation of the eyeball may be used as a point of orientation, as in the so-called physiological methods of localisation. The advocates of these methods claim a greater degree of accuracy with them in the case of foreign bodies far back in the vitreous than with the geometric methods

(5) THE ANGLE OF THE CENTRAL RAY AND OF THE ANTERO-POSTERIOR AXIS OF THE EYE MUST BE KNOWN.—If possible both should coincide. Failing this the angle made by them should be known

(6) THE MAGNIFICATION OF THE IMAGE.—This should be allowed for in the estimations if sufficient to warrant notice. Telerradiograms avoid appreciable

magnifications but a satisfactory compromise is a tube film distance of 36 inches which gives an error of less than 0.5 mm

The various methods of localisation (and they are many) fall into six groups

1 **Physiological Methods**—Several radiograms taken with the eye looking in different directions the tube and the head remaining fixed (*Kohler Dor Belot and Frandet Holz knecht Altschul Grudanski*)

2 **Geometric Methods**—Several radiograms taken from different planes the head and eye remaining fixed (*Sweet McGregor Dixon Ahlborn Ohnishi Muller and Stumpf*)

3 **Stereoscopic Methods** (*Henrard*)

4 **Simple Methods**—Postero anterior and lateral radiograms with various opaque markers (*Arganaraz Wessely Vetter Comberg*)

5 **'Bone free' Methods**—Using small films in the conjunctival sac (*Vogt Landblom Franceschetti*)

6 **Contrast-medium Methods**, in which the globe is outlined by air lipiodol or thorotrast

**Assessment of the Various Methods**—No one method can be said to be the best all have advantages and disadvantages Some methods suit best for certain cases and the choice may depend to some extent on the apparatus available and the mathematic bias of the operator Those who tend to shun an algebraic formula will tend to use the more objective methods

For the most precise localisation the methods of *Sweet McGregor Ahlborn* and *Comberg* are to be recommended The three former require special apparatus In Great Britain and the U.S.A. *Sweet's* method is very popular and *Ahlborn's* on the Continent If no specialised apparatus is available e.g. in war time *Vetter's* method is of value Finally the methods of *Vogt* and *Landblom* are of great use in the case of foreign bodies in the anterior chamber

### Physiological Methods

Observations based upon movement of the suspected shadows are unreliable because (a) if extra ocular but in the capsule of Tenon the muscle the optic nerve or intra orbital fat the shadow may show movement while (b) if intra ocular but near the centre of rotation of the eye the shadow may remain stationary

The more refined methods based on the same principle those of *Koller Dor Altschul Holz knecht* and *Belot* are not now of importance

### Geometric Methods

**Sweet's Method**—In 1898 *Sweet* described his method and first apparatus In 1909 he described his improved apparatus It is remarkable that after thirty years this improved model is still one of the best and most popular methods of localisation (Fig. 694)

A lead ball indicator is used to orientate the centre of the cornea and by

means of a mirror and a telescope this indicator is adjusted exactly 1 cm away from the centre of the cornea and in the optical axis. Two semi lateral radiograms are then taken on one film—the second exposure with a caudal displacement of the tube.

In the Sweet apparatus the tube, ball indicator and film holder are all on a movable stage in constant relationship to one another. The tube, film distance and the angle of the central ray with the optical axis are both constant so that the same indicator therefore serves for both exposures.

The optical axis is fixed by the sound eye, an advantage since often the damaged eye is sightless.

The localisation is carried out in the following six steps:

(1) Place the patient supine with the film on the side of the head nearest the injured eye (Figs 694 (a) and (b)).

(2) Adjust the ball indicator vertically 1 cm above the centre of the cornea.

(3) Take the first radiogram with the tube in the middle position of the scale (the central ray at right angles to the long axis of the patient's body and passing through the centre of the iris of the damaged eye).

(4) Take the second picture with the tube displaced to the end of the scale nearest the feet.

(5) Superimpose the film on the key plate and read the co-ordinates of the two shadows of the foreign body.

(6) Mark these co-ordinates on the chart: their intersections mark the position of the foreign body within or without the eye.

**Method of Adjustment of Optical Axis of the Injured Eye**—R in Fig 695 is the point on the mirror at which light from the indicator ball is reflected into the sound eye (Y). The ball is therefore seen as a virtual image at V. The optical axis of the injured eye (A) will therefore be in the correct line so long as the sound eye is looking at the image of the indicator ball in the mirror.

**Method of Adjustment of Indicator ball**—The operator looks through the aperture (O) in the mirror and aligns the ball indicator over the centre of the cornea of the injured eye. This ensures that the optical axis of that eye is parallel to the film. The ball is then adjusted to a distance 1 cm from the surface of the centre of the cornea by looking through the telescope and bringing the image in the telescope mirror (M) of the cross wire into a position tangential to that of the centre of the cornea.

A small lamp gives the necessary illumination. The variation of distance due to parallax error is stated to be only 0.1 mm.

**The Exposures**—The eyes must remain immobile throughout the two exposures and to facilitate this both are made on different portions of one film (Fig 696).

**FIRST EXPOSURE**—The film is placed *in situ* and the lead shutters opened to leave the centre part of the film unprotected. The tube is set at zero on the scale and the first exposure is made.

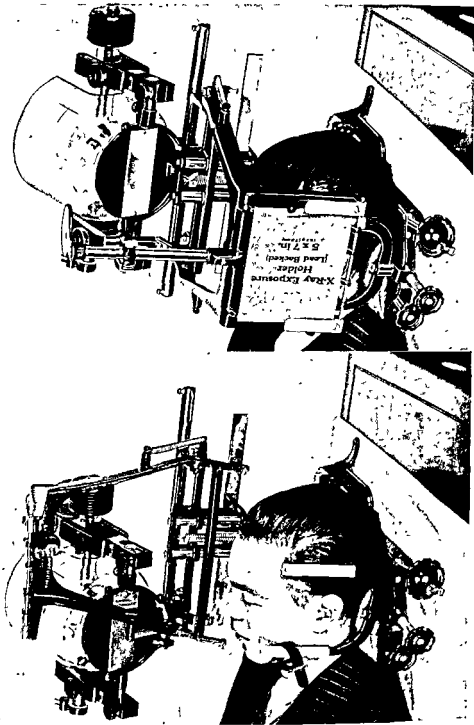


FIG. 694.—Sweet's localiser. (a) The head in position for localising a foreign body in the left eye. (b) The film and tube in position for the first exposure.

**SECOND EXPOSURE**—The tube is moved down to the limit, caudadly, and the upper (cephalic) lead shutter moved down to protect the centre of the film and lay bare the cephalic portion. The second exposure is then made, one 50 per cent heavier being given.

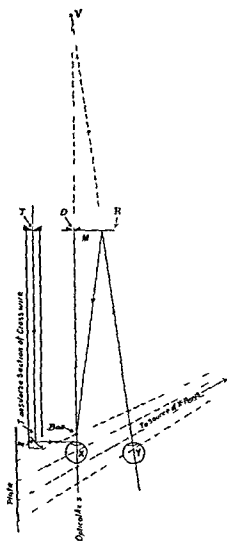


FIG. 695.—Method of adjustment of optical axis of injured eye

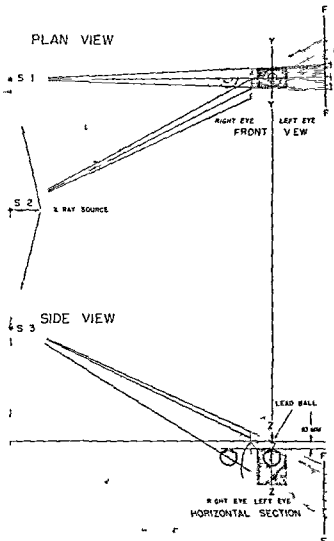


FIG. 696.—Sweet's localisation. Diagram showing the plan and elevation of the two exposures

**Charting the Radiogram**—Place the developed film with its tube side against the key plate (Fig. 697), and move it till the two shadows of the indicator ball coincide with the balls on the key plate.

**C OR D READING**—The film and key are illuminated and the distance of the shadow of the foreign body to the right (C) or left (D) of the indicator ball is

noted and transferred to the corresponding lines of the C or D section of the final chart on the right or left side, according to which eye is being examined (Fig 698)

**E READING**—Without moving the film the E reading (the depth of the foreign body from the tangent of the centre of the cornea) is taken and plotted on the E section of the chart

**A OR B READING**—To take this (the horizontal distance from the vertical zero line in the second radiogram) the shadow of the indicator ball in the

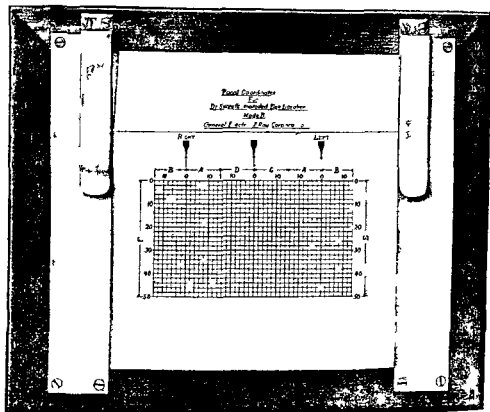


FIG 697—Key plate for Sweet's localisation

second (oblique) radiogram is made to coincide with the right or left indicator ball on the key plate

The A (or B) co-ordinate which crosses the shadow of the foreign body is read and plotted on A or B lines in the chart. (The vertical co-ordinate E should be the same in both readings) If the tube was accurately centred in its holder the shadow of the indicator ball in the radiogram will coincide with those on the key plate and it will then not be necessary to reset the radiogram to read the position of the A and B co-ordinates

bearing cross wires which can be moved back against the closed eyelid. There is a device for marking the quadrants of the eye (6) and a knob in front of the cross wires to facilitate fluoroscopic centring (7).

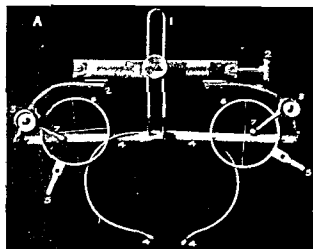


FIG. 699—McGregor eye localiser

the cross wires to facilitate fluoroscopic centring (7).

**Technique**—Place the patient supine on the couch with a support under the neck and fix the head so that the cross wire rings are parallel to the couch top. Ask the patient to look vertically up at a point on the ceiling with his sound eye. This fixes the damaged eye. Adjust the frame till the cross wires are over the centre of the pupil. Screw the ring and cross wire gently back against the closed eyelid of the damaged

eye. By fluoroscopy with the under couch tube adjust the central ray and head till the shadows of the cross wires and knob are superimposed.

**The Exposures**—Lay a dental film on the ring and cross wires in a plane tangential to the centre of the cornea. Make two exposures on the same film the tube displaced first 3 cm. to one side of the centre point and then 3 cm. to the other. Note the tube-film distance which should be not less than 50 cm.

### Charting the Radiogram and Interpretation

1 *To show the Surface Position of the Foreign Body*—Transfer the shadow shift measured on the film to the chart F which is squared in millimetres (Fig. 700). In it  $S^1$  and  $S^2$  represent the shadows and  $T^1$  and  $T^2$  the tube positions. P is the actual position of the foreign body in the plane of the film.

2 *To show the Depth of the Foreign Body*—Measure very accurately with screw dividers the shadow shift in millimetres. Let this be  $s$  mm.

$$\text{Then by the formula } D = \frac{A \times S}{T - S} = \frac{530 \times 2}{60 - 2} = 17 \text{ mm}$$

when A (anode-film distance) = 530 mm. T (tube shift) = 60 mm. S (shadow shift) = 2 mm. and D the depth from the point P on the plane of the film.

3 *To determine if the Foreign Body at this Depth is really Intra-ocular*—Here it must be noted that although the suspected shadow is within the outline of the



the point P<sup>1</sup> is within or without the circle d-e (In the example illustrated it is within (Fig 701) )

If within (as in this case) the exact position in the globe can now be stated as follows

The foreign body is

8 mm  $\frac{\text{above}}{\text{below}}$  the central corneal axis

5 mm to the  $\frac{\text{temporal}}{\text{nasal}}$  side of the central corneal axis

17 mm deep to the plane tangential to the centre of the anterior surface of the cornea

The charts show the foreign body to be  $\frac{\text{internal}}{\text{external}}$  to the outer surface of the eyeball

(Note—In a case where the point P is in the lower half of the elevation view instead of the upper half as shown draw the plan diagram in exactly the same way and in the section cut away *under* the greatest diameter of the eyeball instead of *above* )

**Ahlbom's Method**—*Hugo Ahlbom* has described a procedure which combines the physiological and geometric methods. In it he attempts to avoid the individual pitfalls of the different methods. Thus he eliminates the magnification of the radiographic image by using a tube film distance of 275 metres—takes special precautions to fix the optical axis of the damaged eye in the de-ved line and by the use of an aluminium wedge in the lateral view is able to show the cornea free from other structures and the foreign body in the orbit on one and the same film. For the details of Ahlbom's method the reader is referred to his original paper.

### Stereoscopic Methods

Many workers have attempted to localise intra-ocular foreign bodies stereoscopically. The most systematised method is that of *Henrard*. His routine is as follows

- (1) Preliminary film in *Belot's* position
- (2) Lateral stereograms of the eye the cornea being covered with a contact glass (*coque de Vessely*) and the eye looking straight forward
- (3) Three successive radiograms the tube and head stationary the eye looking respectively up forwards and down

*Henrard's* method thus combines the physiological and geometric methods but neither very accurately

### Simple Methods

**Arganaraz's Method** consists in taking postero anterior and lateral views of the damaged eye the position of which is roughly marked by a wire ring of the same circumference

**Wessely's Method** — *Wessely* uses a contact glass (*coque de Wessely*) with an opaque ring at the site of the limbus. Postero anterior and profile views are taken and the position of the foreign body relative to the glass is clearly seen. What is not apparent however is the relationship of the glass to the eye. This is a source of error. Another difficulty is fitting the contact glass to a damaged eye.

**Velter's Method** — In this method a tiny lead shot is sutured to the upper limit of the cornea and one to the lower (at twelve and six o'clock). Postero anterior and lateral views then show the position of the foreign body relative to the limbus. This is a simple and fairly accurate method and very useful in war conditions.

**Comberg's Method** — In 1927 *Comberg* described a method in which he used as marker a corneal contact glass similar to those used for the correction of ametropia but bearing four opaque dots spaced on the circle of the limbus at angles of 90 degrees.

In this method two views are taken, a postero anterior and a lateral profile.

For the postero anterior view the central ray is first directed vertically downwards on to a small mirror on the couch top, the mirror set at an angle of 45 degrees. The mirror bears a cross line marking. An electric light is placed 2 metres away horizontal with the mirror and in the line of reflection of the central ray axis of the tube. A film is placed *in situ* under the mirror.

The patient is then placed on the couch, the head in the chin nose position. The damaged eye wearing the contact glass has its optical axis centred on the cross lines on the mirror. (This can be seen by the observer from the side.) The central ray and the optical axis thus coincide. The mirror is removed and the exposure made. The lateral or profile view is taken with the patient erect and the central ray in the plane of the limbus of the damaged eye.

The localisation from these two views is very simple. The profile view gives the distance of the foreign body behind the plane of the limbus. On the postero anterior view a line is drawn uniting the two fronto malar sutures. This gives the horizontal line. Two lines are drawn joining the opposing markers of the contact glass. The point of intersection of these two indicates the optical axis. The shadow of the foreign body is joined by a line to this axis point. The distance of the foreign body from the optical axis is then easily measured also to see the angle made by this line with the horizontal.

The various measurements are plotted on to the chart.

### " Bone-free " Methods

These are of value in showing the anterior part of the eye.

*Isgt* and *Landblom* have described simple methods in which a small film is pressed into one point of the conjunctiva and the tube centred on it from the other side. In *Isgt's* two views the film is placed on the nasal side and



FIG. 702.—Dacryoeystography. Stenosis of lower part of nasal duct following an injury which occurred during an antral operation. Note lipiodol outlining the margin of the upper lid.

workers : air by *Staunig* and *Herrenschwan*, *Gasteiger* and *Grauer*, *Spackman*; iodipin by *Farberov* and *Medvedev*; diodrast by *Katz* of Chicago (quoted by *Hartman*). These methods are still more or less in the experimental stage.

### THE LACHRYMAL GLANDS

The lachrymal glands are normally quite invisible in a radiogram. *Schnaudigel* has described a case in which a calcified lachrymal gland was clearly visible in a radiogram. The lachrymal glands may be grossly enlarged in *Mikulicz's disease*, and so cast a soft-tissue shadow.

### THE TEAR DUCTS

The osseous canal may be shown by an

inferior conjunctival sac respectively. *Lindblom* uses the superior conjunctival sac instead of the inferior.

*Franceschetti* has described a method in which he introduces a small film, contained in a sterilised cellophane envelope, into the orbit through an incision in the conjunctiva on the nasal side of the eye.

### Contrast-medium Methods

These are used to outline the posterior part of the globe.

The injection of contrast media into Tenon's space has been tried by a number of



FIG. 703.—Dacryoeystogram of a structure at the junction of sac and duct.

axial radiogram, using an intrabuccal or dental film (*Brunetti Kopplow, Tolh.*).

**Dacryocystography** — The canaliculi, lachrymal sac, and nasal duct may be shown by the injection of lipiodol (*Bollack* in 1924 was the first to use this contrast medium for this investigation since when it has come into general use).

The technique is simple. The syringe is charged with warm lipiodol and the fine cannula is passed into the lower canaliculus. About 4–5 cc is injected and it may be advisable to draw off any fluid or pus in the lachrymal sac before injecting. A little of the lipiodol rubbed along the eyelashes gives a useful orienting mark in a radiogram (Fig. 702).

If the radiograms are taken soon after the injection, the canaliculi should be visible, normally they return some of the lipiodol for a minute or two and longer if the patient keeps the eyes gently closed.

Stereoscopic postero anterior and lateral views should be taken.

The chief use of the method is in the determination of stenosis or obstruction of the nasal duct. If dacryocystorhinostomy is contemplated dacryocystography may give useful information regarding the site of the narrowing and the exact position of the lachrymal sac.

The radiological criteria of obstruction are dilatation of the fundus of the sac, narrowing or obliteration of some part of the duct and failure of the contrast medium to pass into the nasal fossa (Figs. 703 and 704).



FIG. 704 — Dacryocystogram of a stricture at the junction of sac and duct, lateral view.

## REFERENCES

- AYLSON H. *Icta Radiol.*, 1931, VII 212.  
 ALTCHUL, W., *Fortsch. geb. Röntgstr.*, 1922, XXIX, 441.  
 ARGANARAZ R. *Semana med. de Buenos Aires*, 1922, XXX, 62.  
 BELOT, J. and FRAUDET, H. *J. radiol. Paris*, 1916–17, II, 433.  
 BOLLACK J., *Annales d'oeul.*, 1924, CLXI, 321.  
 BRUNETTI L. *Indagine radiologica del canale nasolacrimale mediante clivaggio enorale*.  
*Atti del IX Congresso ital. de rad. med.*, 1930.  
 COMBERG W. *Arch. f. Ophthal.*, 1927, CLVIII, 172.  
 DIXON, G. A., *Ann. r. J. Ophthal.*, 1924, VII, 206.  
 DOR L., *Lyon med.*, 1924, CXXXIII, 172.  
 FAREBROV B. I. and MEDVEDEV N. I. *J. radiol. Paris*, 1930, XIX, 9.  
 FRANCESCHETTI A. *Soc. fran. d'Opt.*, 1934, XLVII, 230.  
 GASTEIGER H. and GRAUER S. *Fortsch. geb. Röntgstr.*, 1929, XL, 272.

- HARTMANN, L., 'La radiographie en ophthalmologie," Paris, 1936  
 HEMARD E., *J belge radiol*, 1927, XVI, 485  
 HOLZKNFCHT, G. *Arch Augenheilk*, 1916, XXXV, 354  
 JUNG Klin *Monat Augenheilk*, 1922, LXVIII, 385  
 KOHLER, A., *Fortsch geb Rgtstr*, 1902, VI, 190  
 KOPYLOW M B, *Rontgenpraxis*, 1930, II, 686  
 LINDBLOW K, *Acta Radiol*, 1934 XV, 615  
 MCGRICOR D B, *Brit J Radiol*, Nov 1939, Vol XII, No 140  
 ROLLET MALOT and AURAND, *Rev gen d opht*, 1913, XXXII, 222  
 STAUNIG, K. and von HERRENSCHWAND F, *Fortsch geb Rgtstr*, 1927, XXXVI 372  
 STUMPF P *Munch med Wschr*, 1916 LXIII, 1606  
 SWEET W M, *Amer J med sci*, 1898 CXVI, 320  
 TOOKF F T *Brit J Ophthal* 1934 XVIII 695  
 TOTZ Z *Klin Monat Augenheilk* 1933, LCI, 390  
 VETTER M *Bull Soc d Opht de Paris* 1919 XXXII, 153  
 VOGT, A *Schweiz med Wschr*, 1921, VII 145

*PART SIX*  
CINERADIOGRAPHY

BY

RUSSELL J REYNOLDS CBE, MB, BS FRCP,  
FRF, DMRE

## *PART SIX*

### CINERADIOGRAPHY

#### CHAPTER LIX

#### INTRODUCTION

EVER SINCE the discovery of X rays by Professor *Rontgen* in 1895, radiologists have been attempting to reproduce photographically the moving shadows seen on the fluorescent screen

The principle of the ordinary cinematograph was discovered in 1896, and the manifest advantages of this, could it be combined with X rays, was quickly apparent

In 1897 *John MacIntyre*, of Glasgow, showed a film illustrating the movements of the bones of a frog's leg to the Glasgow Philosophical Society. The movement he showed was lifelike and informative, but the picture was of course, synthetic. He had taken a number of ordinary isolated radiograms of the leg in different positions, and then, arranging them in order had transferred them to a cinematograph film. This was an extraordinary feat, considering the primitive and feeble apparatus in use at that time, and testifies to *MacIntyre's* ingenuity and patience

#### METHODS OF PRODUCTION OF CINERADIOGRAPHIC FILMS

So far there would appear to be only three ways by which an X ray cinematographic effect may be obtained

The Synthetic Method, briefly described above

The Direct Method, in which a series of skigrams is taken at short intervals upon a long strip of film

The Indirect Method, in which a photograph is taken of the image appearing on a fluorescent screen

Before describing these in greater detail, something must be said of the various early workers in this subject and the methods which they employed.

#### EARLY HISTORY OF CINERADIOGRAPHY

In 1898 *Roux* and *Balthazard* made a number of strips each containing 12 serial pictures 3 cm wide by 6.25 cm long, these were exposed directly for a second each at regular intervals of 10 seconds, and represented the

movements of the stomach of rats and frogs. About 1901 *Carallo* took a number of films of small objects and parts at a rate of 5 to the second using a mechanism much like that of the ordinary cinematograph camera. He used exposures at a 1 second interval to record very slow movement. In 1902 *Lety Dorn* showed a synthetic film of the movements of the knee and elbow joints obtained by placing in order a number of pictures taken at different times of different positions of the bones. In 1907 *Kohler* made by a similar method a film showing the movements of the thorax in breathing. In 1909 *Kaestle Rieder* and *Posenthal* made several serial films of stomach movements on a rather large film with a 1 second interval between exposures. In 1911 *Haenisch* devised an apparatus by which he could expose 10 plates at short intervals by fixing them on a wheel which brought them one after another into position in the beam of X rays. *Poehler* about the same time made an ingenious apparatus by which 60 films—each in a holder—were held close together in a row and pushed forward so that each fell on its face immediately it was exposed. *L. G. Cole* may also be mentioned among well known early workers.

*Jarre* and *Cumming* describe the Cinex camera which they use at the Grace Hospital in Detroit for studying the kidney ureter and bladder. They use films 3 inches wide when investigating one side of the body only and 10 inches to 12 inches for both sides. Their pictures at any rate in 1930 were perhaps not truly cinematographic for the interval was between 1/2 and 1 second but this speed is perfectly effective for a good many movements.

*Macrez* and his colleagues *Gianturco* and *Little* at the Mayo Clinic in America have investigated the workings of the stomach and intestines of animals with a direct apparatus which will take 4 to 6 pictures a second each 4 inches by 3 inches on 30 foot strips of film. One must also mention *Clanberlain* of Philadelphia who produced some films of the heart by the direct method in 1926.

The above is not by any means a complete list of those who have done pioneer work in this subject but includes most of those whose work is well known.

Among the more recent workers mention must be made of *J. van de Muele* of Brussels of whose work more will be said later and *A. E. Barclay* who has also constructed a camera for use with the direct method.

Working with the indirect method along the same lines as the author are *Janter* of Bonn who has produced many films both of animals and human subjects. *Stewart* of New York. *Dryan* of Paris and *Putti* in Italy.



## CHAPTER LX

### METHODS OF CINEPADIOGRAPHY

#### THE SYNTHETIC METHOD

This was the original method employed by *MacIntyre* and by several of the early workers who came after him. While it can be made to give a lifelike representation of movement under certain conditions its limitations are so great that it is in its original form no longer employed.

A modification of it is however still used by *Van de Maele* and some other workers in which a series of isolated films is rapidly taken the movement being carried on the whole time. A further note about this will be found under the direct method.

This modification can hardly be compared to *MacIntyre's* original process where a film was taken the object to be radiographed being held still as long as was required then after one exposure being moved to a fresh position ready for the next. Then when the series had finally been completed there remained the problem of transferring it to a standard sized film for projection. It was moreover quite useless for getting representations of involuntary movements such as those of the alimentary canal which could not be kept stationary while the exposure was made.

#### THE DIRECT METHOD

In its essentials this method consists in passing continually into the position normally occupied by the viewing screen or the cassette a long strip of film the dimensions of which will vary with the size of the field which it is desired to cover. The film is clamped between two intensifying screens and the rays switched on so that the film is exposed in the usual manner. As soon as the exposure is over the film moves along and a second picture is taken in the same way.

The essential features of such an apparatus are that there should be some method of passing such a large area of film into position and holding it absolutely still while the exposure is made, and then of carrying it through the various developing processes. These requirements are not easily fulfilled and present many serious mechanical problems. In order as far as possible to minimise the difficulties of designing the apparatus a restricted field of 4 inches by 5 inches is usually employed but this sets a limit to the parts of the body which can be examined.

It is of course necessary as in the synthetic method to reproduce the serial skiagrams for projection purposes on a standard size (16 mm or 35 mm) cinematograph film. In this connection it must be remembered that if a smooth movement is to be obtained the film must pass through the projector at a minimum speed of 12 frames per second.

If it is not possible to take the serial skiagrams at a sufficiently rapid rate owing to the thickness of the part to be radiographed or to the mechanical difficulties involved each skiagram of the series may be printed two or three times in succession on the projection film and the result is in this way stretched so that continuity without undue rapidity is obtained.

However many of these difficulties have been minimised. *Van de Maele* of Brussels has in particular been successful with this method.

The following is a brief description of his apparatus and an account of his technique serves well to show the modern development of the direct method of cineradiography.

He uses Gevaert film coated with ordinary double emulsion and the dimensions of each frame are 120 mm long and 178 mm wide (approximately 4.8 inches by 6.1 inches). The edges of the film are perforated at 2 cm intervals with rounded holes; these have shown no tendency to tear even after the film has been run through the apparatus 10 times. The problems of development are difficult because the ordinary cinematographic technique cannot be used on account of the double emulsion. The film is developed in a special tank in which it is wound over a frame fitted with teeth which engage in its perforations. This worker uses a 90 kv Philips Rotax tube which has a rotating anode in order the better to stand up to heavy currents with a small focal spot. The tube and its leads are protected by a shockproof casing. The equipment has been run habitually at 80 kv with a current of 80 ma for exposures of over 20 seconds; with a lesser load the exposure time has been lengthened to 30 seconds but the tube has so far shown no signs of wear. It is fed by a 4 valve generator in a Graetz mounting which does not supply a continuous current but the output curve is considerably flattened by the use of long shockproof cables having a considerable capacity. There is no fear of over irradiating the patient for in 20 seconds he only receives a dose of 38 r which is equivalent to about one ninth of the skin unit dose or that quantity of X rays which will cause a moderate reddening of the skin. This economy of radiation is obtained by the use of an obturator which is ingeniously synchronised with the film and cuts off the rays while the frames are being traversed. For making records of the movements of abdominal organs a rotary grid of the Siemens type is employed; this rotates at 90 r.p.m. The function of a grid is to improve detail. As this necessarily interrupts a large part of the radiation it is not possible to take good pictures faster than 13 frames per second. The apparatus is driven by a 1 h.p. motor operated by 3 phase alternating current. It will run smoothly and continu-

ously at a rate of 16 frames a second. This admirable result is attained by perfect synchronisation—a positive drive to the film—a constant band of slack on either side of the propelling apparatus—and a spring device by which the reinforcing screens grip the film smoothly at every exposure. For the purpose of projection the large negative is reduced to a 35 mm. film.

A modification of this apparatus which is still in its final stages of development overcomes the difficulty associated with the rapid traverse of a heavy band of film by using a pack of single films operated by a rotary feeder with a continuous movement. The advantage of this principle over the ordinary one by which the film has to be stopped and restarted 16 times in a second is obvious. The frames measure 12 cm. by 14 cm. and are exposed at the rate of 18 per second. The negatives are reproduced in succession on ordinary cinematograph film for projection.

*Van de Maele* claims to have solved the formidable mechanical problems of the direct method. He now calls for improved intensifying screens and more sensitive film to enable him to improve his results still further.

### THE INDIRECT METHOD

**Principle of the Apparatus**—This differs essentially from either of the methods previously described for whereas in their case skiagrams were obtained which had afterwards to be reproduced photographically for projection here the image is obtained directly on the photographic film by the utilisation of the light from the fluorescent screen.

In order that a satisfactory result may be obtained it is necessary to have

(1) A sufficiently brilliant screen image so that it may impress itself on a cinematograph film exposed for a small fraction of a second.

This requires

- (a) An X ray generator of fairly high power
- (b) An X ray tube that will take heavy currents
- (c) A lens which will give fine definition with wide aperture
- (d) A cinematograph film as sensitive as possible to the light emitted by the screen

(2) Protection of the film in the camera from the direct radiation passing through the fluorescent screen

(3) Reduction as far as possible of the load on the tube

(4) Protection for the patient so that he may not receive an excessive dose of rays this being the most important of all

The following is a description of the indirect apparatus which has been evolved by the author and of his technique at the present time.

**General Description of the Russell Reynolds Apparatus**—The radiographic and camera units are entirely separate from one another but both are mounted

on the same set of rails to keep them in line and allow of easy adjustment of the distance between them. Their controls are taken to a common switch table and by means of a single switch the camera unit can be cut out of circuit and the radiographic unit may be used by itself for the production of ordinary skiagrams.

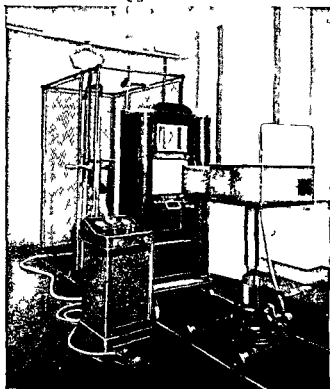


FIG. 65. Russell Reynolds cineradiographic apparatus.

light available it is fitted with a Zeiss 0.85 lens. The film chosen is specially sensitised to the particular wave length of light emitted by the screen employed.

**Protection of the Film in the Camera from the Direct Beam of X rays**—The camera is placed in front of the fluorescent screen and separated from it by a distance of 63 inches. This is an arbitrary distance but was chosen because at this distance a convenient sized field of 12 inches by 15 inches was covered on the 16 mm film.

The sheet of lead glass usually placed in front of the screen to obstruct the further passage of the rays cannot be used because it absorbs at the same time about a quarter of the visible light from the screen and this is a fraction which one cannot afford to lose in motion picture work.

There is thus a fairly powerful beam of X rays reaching the camera unit

ordinary skiagrams

#### The Radiographic Unit

—This follows normal practice and consists of

(1) A vertical screening stand of the usual type for examining patients (Fig. 70a) fitted with an alternative fluorescent screen which is fully protected.

(2) A rotating table upon which the patient can stand.

(3) A 10 kw. Metalix tube of the heavy anode type fitted to the stand with the usual diaphragms.

(4) A 151 v. transformer.

**The Camera Unit**—For reasons of economy and ease of handling a standard 16 mm. Victor camera is used (Fig. 70c).

To make the most of the

and so the latter is enclosed in a wooden box lined with 3 mm of lead which protects against both direct and scattered radiation.

An aperture is left in the front of the box which just fits the lens and it is found that the great thickness of the glass in the combination is sufficient to protect the film as it passes the gate at the moment of exposure.

#### Protection of the Patient

The rays from the tube are filtered through 0.5 mm Al to intercept the soft radiations as the current through the tube may be as great as 60 ma at a maximum of 120 kv. The distance of the anticathode to the screen varies. As exposures up to 10 seconds may be required to obtain a record of a complete cycle of movement the patient is protected and the strain on the tube relieved by means of a synchronising switch.

It is worth recording that even with a 10 second exposure and the maximum current when the switch is in use the total dose received by the patient is under 30 r and the author has given a total exposure of 30 seconds and repeated this in a few days to the same area without producing any ill-effect at all on the patient.

**The Synchronising Switch**—The following is a brief description of the synchronising switch.

The camera takes "pictures" for every revolution of the driving spindle and the shutter opens and closes "times" uniformly during each revolution. The intervals of time during which the shutter is open and closed respectively are equal. The length of the exposure therefore can be exactly regulated by varying the speed of the driving spindle. The function of the synchronising switch which is placed in circuit with the primary of the transformer is to

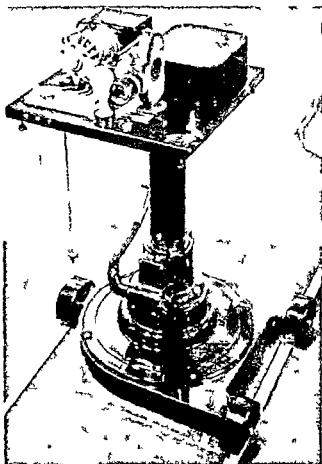


FIG. 70. Revnol cineradiographic camera unit.

switch on the tube during the time the shutter is open and to switch it off when the shutter is closed and the film is being transported. This ensures that the patient shall only receive half the radiation he would receive if the tube were switched on for the whole time and the load on the tube is correspondingly reduced.

The switch itself is made of a flat disc of Keramot fitted with appropriate copper contacts and rotating in an oil bath.

**General Description of Camera Unit**—The component parts of the camera unit are mounted on a metal platform which is carried on a vertical ram so

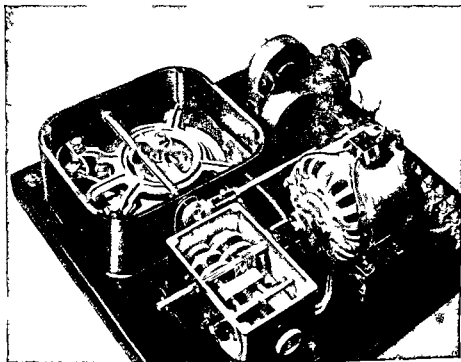


Fig. 70 Synchro switch of the Russell Reynolds apparatus.

that its height can be adjusted by the operation of a foot pedal connected with an oil pump. The driving shaft of the camera is rotated by a small synchronous motor operated through a gearbox with an adjustable range of speeds as shown in Fig. 707. The shaft is also coupled to a rotating contact breaker which makes and breaks the primary circuit of the X-ray generator simultaneously with the opening and closing of the camera shutter. As the motor is completely synchronous and the speeds provided by the gearbox are always multiples or sub multiples of the frequency of the A.C. supply the camera shutter opens not only in synchronism with the

circuit breaker which indirectly controls the high tension supply to the X ray tube but also in complete synchronism with the A C impulses of the primary circuit. In this way the period of time during which each individual frame is exposed to the light from the screen corresponds exactly to a given number of A C impulses. Thus ensures that each frame receives the same exposure. Fig 708 illustrates what happens when the camera is operated at synchronous speeds and also what may happen when a non synchronous speed—e.g. 30 frames a second—is selected.

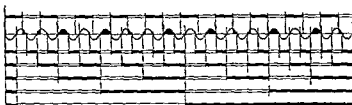


Fig 708.—Diagram of the synchronism of camera shutter and circuit breaker at different exposure speeds.

One of the major problems encountered in designing the gearbox was that of ensuring that no neutral or free position was possible when changing from one speed to another. If this happened the camera shutter or circuit breaker might be inadvertently turned, the relationship between the alteration of the primary supply and the opening and closing of the camera shutter might be disturbed and the timing might be thrown out of phase. The shutter might still operate synchronously but would be out of phase with the voltage supply of the tube. When working at 50 frames a second it might well be just so much out of phase that no energy would reach the fluorescent screen during the period when the camera shutter was open. As the unit is designed to operate on a 50 cycle supply the frame speeds selected are 50, 25, 12½, 10½, and 3½ frames per second—all sub multiples of 50. The control which changes gear simultaneously operates a pointer which indicates the corresponding frame speed.

Complete synchronisation is maintained by a number of keys inside the gearbox which slide in keyways in the main driving wheels and ensure that the drive to the camera is not taken up until the relation of the key to the leverway in its corresponding wheels is exactly correct.

The whole of the switching arrangements for the camera unit are included in the control table for the X ray transformer unit. They are so arranged that when the control switch on the table is turned on, the driving motor is automatically started and runs for a predetermined number of seconds, after which it is automatically switched off. The control table also operates a contactor by means of a simple switch, to short circuit the circuit breaker and interrupt the supply to the synchronous motor in order that the equipment may be used for ordinary routine radiography. The cover of the camera unit incorporates a source of red radiation directed towards the fluorescent screen in order to "kill" after glow. The camera lens is shielded by means of a long

hood to prevent any reflected light from reaching the front surface of the lens. A door in the wooden cover allows ready access to the camera which can be easily removed for reloading and adjustment.

The base of the camera can be rotated through a complete circle in order that the camera may be employed if desired in conjunction with more than one radiographic apparatus.

**Size of the Field**—The standard sized field for normal work is 12 inches by 15 inches and this is large enough to allow the whole thorax to be included but the apparatus can be used to take pictures of any dimensions smaller than this with consequent increase in detail.

In fact a field 4 inches by 5 inches is frequently employed but no sizes other than these for the extremely accurate focus required makes it undesirable to make too frequent alterations in the position of the lens.

**Band Projection**—It is only necessary to expose a sufficient length of film to obtain one complete cycle of any movement desired. The negative thus obtained is printed continuously on to a standard positive film and a short length say 3 feet of this again joined in a band may be passed through the projector indefinitely.

**Comparison of the Direct and Indirect Methods**—It is difficult to compare the direct and indirect methods. Not only do they differ essentially in principle but the type of result they produce varies also. They should be regarded as complementary developments in radiological progress.

When attempting to compare the various methods we need concern ourselves no longer with the first or *Synthetic method* in its original form. It was at best only a temporary makeshift which was made necessary by the elementary equipment then available.

*The Direct method* is expensive both as regards the film and the apparatus and it is associated with many mechanical problems.

The results obtained should be a series of rapid serial skiagrams and show detail as good as those produced in the usual way. They should therefore be suitable for the consideration of slight movements in small areas such as the barium washed rugæ of the alimentary canal or the detailed movements of the pylorus.

Moreover the apparatus is most likely to be a success in a research institution where a large amount both of time and money is available and the special methods of handling, developing, fixing and photographing on to a film suitable for projection can be adequately carried out.

*The Indirect method* on the other hand is cheaper and more easily managed. The apparatus is best described as a straightforward X ray set with a special cinematograph attachment. A word of warning is needed here. It is not possible to place the camera unit in front of any X ray set and expect to get a result. The handling of the film and even the printing of short lengths suit



able for projection as a hand can be carried out either in a private house or in a hospital

It is the method suitable for studying the functions of organs, and at the present time for their minute structural detail it should be combined with ordinary still skiagrams which can be taken with the same apparatus

### FUTURE OF CINERADIOGRAPHY

It must be remembered when discussing the future of cineradiography that as yet there has been little time to do more than develop an apparatus that will produce satisfactory results

The advantages obtained from a permanent moving record of the organs of the body are obvious not only for the clinician but for workers in the allied medical sciences also. Until recently radiology has been limited to a static rendering of shadows with the exception of a screen examination which is memorised and interpreted by the radiologist. Consequently diagnoses have had to be made on films taken at a particular instant take as an example the sign of persistent filling defect in carcinoma of the stomach. Such diagnostic criteria will always be fundamental but it is not too much to hope that we may now go a step farther and make diagnoses on function also. The persistent deformity will remain but the movements of the stomach and its response to standard technique will vary from the normal.

It will take time before these additional methods of diagnosis are fully worked out. The possible shapes and positions of any moving organ are almost innumerable and until we can safely state the limits of the normal we cannot attempt to diagnose what is pathological.

Much work must be done on all parts of the body such as the heart lungs alimentary canal renal tract gall bladder and joints.

As this form of examination is improved and comes into greater prominence, so also will its applications increase. Radiology itself developed gradually and cineradiography must necessarily do the same.

We must first work out a moving radiographic physiology a moving radiographic pathology will follow.

In the study of the normal and abnormal alike cineradiography may be in its turn much enriched by the use of the sound track to combine with the photographic record a synchronised record of audible phenomena such as the heart sounds breath sounds speech and sounds produced by abnormal accumulations of fluid. For the study of the heart in particular the film may usefully incorporate a tracing of the electro cardiographic record and possibly also of the record made by the 'densograph' or ionograph an apparatus which gives a continuous measurement of the quantity of X-rays passing through any given part of the heart and therefore of the thickness of the heart muscle at that point at any particular instant. The chief component

of the ionograph is an ionisation chamber filled with methyl bromide. The instrument gives valuable information when used in conjunction with the electro cardiograph.

Unless there is a radical and far reaching change in the method of production of X rays necessitating corresponding changes in the design of normal equipment it is unlikely that the methods previously described for obtaining cineradiographic pictures will be altered except by improvement in details.

Workers in both the direct and indirect methods have this in common—they require even faster screens with the finest grain.

Those interested in the indirect method need in addition a faster fine grain cinematograph film and a lens with a greater aperture even than 0.8, provided that definition can be maintained.

The next ten years will see great advances in cineradiography. It is not too much to say that before long physicians and surgeons may think as little of asking for a cineradiogram as they do now of asking for a normal radiogram and that cineradiography will take its place as a routine procedure in suitable cases.

## CHAPTER XVI

### CLINICAL APPLICATIONS OF CINERADIOGRAPHY

#### EXAMINATION OF THE THORAX

WHEN a cinematograph film is made of the thorax for the study of the lungs or cardiovascular system practically the same routine is carried out as regards the positioning of the patient as when an ordinary X-ray examination is made. The essential difference lies in the fact that the patient is required to move during the taking of the film.

Exposures are made at the rate of 12 per second for normal adults and even faster (20 per second) in the case of thin adults and children. It follows that it is always possible to reproduce movements at the same speed as that at which they took place and when films can be taken at the faster rate a slow motion effect is obtained.

When viewing the film for diagnostic purposes it is advisable to project it on to a screen about the same size as the fluorescent screen photographed. The shadows then appear the normal size.

A slight degree of distortion of the true outline occurs of the same nature and degree as that present in any radiographic examination in which the screen-anode distance is short (36 inches) and to obtain the true size of the heart an orthodiagram or teloradiogram may be made with the same apparatus the cinematograph attachment being cut out of circuit for the time being.

As this method is especially designed to study the movements of organs slight distortion is of little importance.

#### EXAMINATION OF THE HEART

When making a full cineradiographic examination of the heart the customary positions antero-posterior first and second oblique are normally employed together with any special position which may be thought advisable in any particular case.

Here even more than in a plain radiographic examination must the technique be varied with the condition of the patient for in addition to his position it is necessary to consider whether he should breathe normally and quietly, whether he should remain with breath held in either full inspiration or expiration or whether one very deep breath should be taken during each exposure.

In general however the following four films are made.

(1) With the patient rotating slowly from the first to the second oblique positions.

- (2) Antero posterior with the breath held in full inspiration
- (3) Antero posterior with the patient breathing quietly throughout
- (4) First oblique with the patient breathing quietly

(1) gives a good general impression of the whole thorax for the intrathoracic shadows. As they pass in relation to one another a remarkable almost stereoscopic effect is produced.

(2) The aeration of the lungs increases the contrast and perhaps allows of an increased speed in making the exposure. It also clears the surrounding structures away from the heart and opens the intercostal spaces.

(3) and (4) These are perhaps the most valuable of all as they show the normal condition of the heart at rest.

These four exposures may be taken at one session any or all of which may be of 10 seconds in duration. Although one is still well within the limit of dose it is advisable to wait until a later occasion for further examination to enable the result to be studied and an opinion formed as to what further films are desirable.

Further information may be afforded by

- (1) A close up view of any particular area
- (2) A barium swallow to outline the left auricle
- (3) A general view of the condition of the heart after exercise

On examining the film of a heart one can take note of the following points

- (1) The shape size and position of the heart
- (2) Is there any arrhythmia or any irregularity in the auricular or ventricular pulsation?

(3) Is the pericardium free at the cardio-phrenic angles on deep inspiration?

(4) The action of the heart in the act of sneezing or coughing

(5) The action of the heart after violent exercise.

In pathological conditions one may also study the size shape and position or any calcification in the shadow of the aorta the heart's action in auricular fibrillation and other lesions where the beat is arrhythmic. In some cases of pericarditis with effusion the heart may be seen beating within the pericardial cavity and the way in which the pericardial adhesions when present limit the movements of the pericardium may be studied also the curious piston-like action of the heart which is seen in many cases of double artificial pneumothorax.

A permanent record can always be kept for comparison with any future records made.

## EXAMINATION OF THE LUNGS

When examining the lungs the films are taken at the same rate as in the case of the heart—12 to 25 per second but the variation in the technique is greater. It is however convenient to start with the following three positions

- (1) Rotation from first to second oblique positions with the breath held in full inspiration
- (2) Postero anterior with the patient breathing normally
- (3) Postero anterior with the patient taking as deep a breath as possible during the exposure

In the normal chest the regularity of the broncho vascular tree the degree of translucency of the lungs which increases uniformly with inspiration will be noted together with the type of breathing whether thoracic or abdominal and all the usual points of which mental note is made during the ordinary short screening examination

With the breath held in cases of artificial or spontaneous pneumothorax if the patient is rotated any adhesions present will show up clearly and their position can be determined

The extent of collapse can be appreciated with any variation in the position of the heart and mediastinum When the patient is breathing on deep inspiration there is frequently a considerable lateral movement of the heart and mediastinum and expansion of the collapsed lung Should a cavity be present its exact condition is revealed

The degree of asymmetry in unilateral paralysis of the diaphragm may be noted In cases of phrenic avulsion or crush the diaphragm may show paradoxical movement

When the patient moves the fluid in a hydro-pneumothorax cavity is seen to splash

Particularly valuable information is obtained in cases of bronchiectasis after a lipiodol injection into the bronchial tree The patient may be allowed to cough during the exposure the effect of this coughing on the dilated bronchioles shows up well

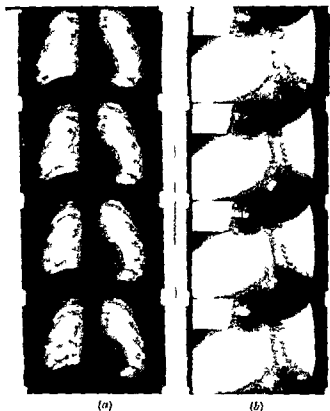


FIG 9—Four frames of (a) a thorax and (b) a bar in meal being swallowed

All these movements can then be studied at leisure from the bands of film made afterwards

### EXAMINATION OF THE ALIMENTARY TRACT

Cineradiographic examination of the upper part of the alimentary tract has been carried out by the author systematically. The results formed the subject of a Hunterian Lecture delivered by the author in 1937

The movements which have been studied are those of

- (1) Mastication
- (2) Swallowing of solid and liquid food
- (3) Filling of the stomach
- (4) Normal stomach movements
- (5) Passage of the meal through the pylorus and duodenum

### DEGLUTITION IN THE NECK

Liquid food is projected to the back of the mouth by means of an upward and backward movement of the tongue. The stimulus from this sensitive area causes the suprahyoid and associated muscles to draw up the hyoid and the rest of the pharynx which is attached to it. The hyoid is drawn upward backward and rotated about a lateral axis and the food is then jerked into the pharynx which is waiting to receive it. This jerk probably performed by the muscles of the back of the tongue carries the bolus over the epiglottis.

The liquid then passes rapidly down clinging to the sides of the œsophagus which seems to be dilated to receive it. It does this under the influence of gravity and not by means of a peristaltic wave.

On reaching the thoracic inlet the food is held up by a constriction at the level of the aortic arch. It collects here in the form of a cone until it is allowed to pass into the lower part of the œsophagus.

Solid and semi solid food behaves in the same way until it reaches the aortic arch.

### DEGLUTITION IN THE THORAX

After the bolus has reached the level of the aortic arch it may act in one of three ways

(1) The food may pass down continuously under the influence of gravity in a thin stream and be held up at the cardiac orifice by the contraction of that sphincter. In this case the œsophagus fills up like a test tube until the pressure of fluid is sufficient to open the cardia when all the food enters the stomach at once through the narrowed orifice in the diaphragm. The barium passes down the posterior wall and has a horizontal upper level.

(2) It may pass down as in the previous case but with the addition at intervals of a gush of fluid which passing rapidly down the œsophagus under

the influence of gravity enters the stomach directly without any delay at the cardia

(3) The liquid may pass into the stomach in a constant thin stream under the influence of gravity. In such a case there is no delay at the cardiac orifice and the liquid passes straight into the stomach. It appears to pass down the oesophagus mainly along the posterior wall.

Solid food is held up at the level of the aortic arch and afterwards it passes slowly down the oesophagus not under the influence of gravity but by peristalsis. It passes down very much more slowly than is the case with liquid food. When it reaches the cardia it passes straight through as the wave of peristaltic relaxation reaches the sphincter.

Semi solid food which is too thick to flow easily and yet is still liquid passes down by peristalsis in the same way. Here again it passes into the stomach without any delay.

### FILLING OF THE STOMACH

The food enters the stomach through the comparatively narrow cardiac orifice. It first passes into the upper passive part of the stomach which comprises most of the fundus.

No peristaltic contraction occurs here and the organ merely dilates under the normal muscular tone as more food enters it.

When the pressure of food in it reaches a given amount the opaque emulsion falls into the second portion.

As soon as the second part of the stomach is distended with food peristalsis commences and the food is passed along to the third or pyloric part.

The contraction of the muscle during peristalsis causes a rise in pressure in the second part and the food taking the path of least resistance is momentarily regurgitated into the first part.

The remainder of the food is carried along to the third part by which it is forced by general contraction into the duodenum.

All this takes place very rapidly in a stomach which has good tone. In one of the films the emulsion was passing through the duodenum within 20 seconds of being swallowed by the mouth and the barium can be seen passing into the jejunum while the rest of it is still entering the stomach or passing from the first part of that organ to the second.

### MOVEMENTS OF THE NORMAL STOMACH

The normal stomach may be broadly divided into two main types—the *tonic* and the *hypotonic*.

The first is vigorous in its action and lies high up in the abdomen. The second lies low in the abdomen and is sluggish in its action.

The stomach itself can be divided into three parts which are best studied in the tonic and active type

The first part is passive acting only as a reservoir for the food

In the second part movement takes place and is of a peristaltic nature commencing as soon as it is distended with food. The peristaltic wave carries the food into the third part where a general contraction takes place which forces the meal through the narrow pylorus into the duodenal cap.

In the hypotonic stomach these three stages are not so well shown but the generalised contraction of the third part seems to be invariable as also is the passivity of the first. Whether the second part undergoes peristalsis is not so clear.

It is possible that the change in character of the movement depends upon the different arrangements of the muscular coats of the various parts of the stomach. In the first part of the stomach there are many oblique fibres and few circular. In the second part the circular fibres are more numerous and it is here that peristalsis starts while in the third the circular coat is very thick and some of the longitudinal fibres are interlacing with it. This would allow of the generalised contraction already noted.

It will have been noticed that the passage of food through the duodenum does not synchronise with the time of its escape from the stomach. This may be due to the fact that the circular muscle fibres of the stomach are not continuous with those of the duodenum and only a few longitudinal fibres which are not so concerned with peristalsis pass between the two organs.

### EFFECT OF DELAY ON NORMAL STOMACH

Both tonic and hypotonic stomachs contract vigorously when they are suddenly distended with a large quantity of food. This is presumably because the muscle fibres are stretched and react by contracting.

After a short while the fibres lengthen and the organ becomes adapted to the large bulk of food. The stretching of the fibres no longer occurs so the stimulus to violent contraction is no longer present.

The most active part of both types of stomach and the part that goes on acting longest is the pyloric end but even this becomes less vigorous and finally does not force enough barium through the pylorus to cast a shadow.

Even early in digestion the second part of the hypotonic stomach becomes quiescent. As the food now remains longer in the stomach it can be better mixed with the gastric juice but even so in the tonic stomach 20 minutes after food has been taken it is still passing rapidly through the duodenum.

### EXAMINATION OF JOINTS

A cineradiographic examination is of great value in the study of joint movements. Specified movements of all kinds may be carefully examined in health and disease.



Using this method of examination it is interesting to note that the maximum movement in flexion and extension of the cervical spine takes place between the fourth and fifth and fifth and sixth cervical vertebrae.

In cases of trauma in the cervical region displacement of a vertebral body and limitations of movement in individual joints due to the presence of adhesions have been diagnosed.

The locking movement which takes place in the normal knee joint on extension of the leg has been studied.

It is perhaps particularly in the field of orthopaedics that the close up examination of a limited area of 4 inches by 5 inches is of value. This method gives a bigger image with finer detail and its value is exemplified by studying the condition of the intercarpal joints.

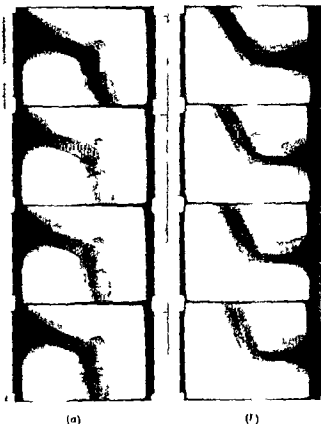


FIG. 710—Four frames of (a) a knee and (b) an intercarpal joint during movement.

### CONCLUSION

What has been stated above is little more than a brief summary of the more important applications of cineradiography. The close up method can be used for examination of the gall bladder or pelvis of the kidney etc. just as is done in the direct method of cinematography.

A cineradiographic investigation enables one to obtain a rapid inexpensive and permanent record of the functioning of active organs and moving joints. The continuous bands of film enable one to study movements for an indefinite period which has been impossible heretofore.

The recording films may be used for (a) purely diagnostic purposes, (b) comparison with former records to study the effects of treatment or the progress of a pathological condition, (c) teaching purposes, (d) transmission abroad or elsewhere either for the purpose of obtaining specialists' opinions.

on the nature of the case or for information as to the condition of the patient in the past

Cineradiography is still in its infancy. So far there has not yet been time to make anything like a complete study of its advantages and possibilities in any one of the directions indicated, quite apart from the vast field of work covered by modern radiology

### BIBLIOGRAPHY

- BARCLAY, A. E., *British Journal of Radiology*, 1935, VIII, 652  
 CARVALLO, 1901 (quoted by Van de Maele—see below)  
 CHAMBERLAIN, W. E., *Radiology*, 1926, VII, 185  
 COLE, L. G., *Arch. und Atlas du normalen und pathologischen Anatomie in typischen Röntgen*, Leipzig, 1912  
 GLANTURCO, C., and ALVAREZ, W. C., *Proceedings of Staff Meetings of the Mayo Clinic*, 1932, VII, 669  
 GOETT, T., and ROSENTHAL, J., *Munchener medizinische Wochenschrift*, 1912, LIX, 2033  
 GOTTHEIMER, V., and ZWIRNER, E., *Fortschritte auf d. Geb. der Röntgenstrahlen*, 1933 XLVII, 455  
 GROEDEL, H., and FRANKE, H., *Fortschritte auf d. Geb. der Röntgenstrahlen*, 1933 XLXIII, 65  
 GROEDL, T., and GROEDEL, F., *Deutsches Archiv für klinische Medizin*, 1913, CVIII, 52  
 HAENISCH, F., *Ibid.*, 1911-12, XVI, 354  
 HIRSCH, I. S., *Ibid.*, 1934, VII, 728  
 JACOBI, J., *Verhandlung der Deutschen Gesellschaft für Inner Medizin*, 1933, XLV, 229  
 JACOBI, J., JANKER, R., and SCHMITZ, W., *Deutsches Archiv für klinische Medizin*, 1931-32, CLXXII, 497  
 JANKER, R., *Deutsche medizinische Wochenschrift*, 1932, LVIII, 1094  
 JARRE, H. A., and CUMMING, R. E., 1930, *Journal of Urology*, 1930, XXIV, 423  
 KAESTLE, K., RIEDER, H., and ROSENTHAL, J., *Archives of the Roentgen Ray*, 1910, XI, 3  
 KNOX, R., *British Journal of Radiology*, 1925, XXI, 142  
 KOHLER, A., *Ibid.*, 1907, XI, 218 and 292  
 LEVY DORN, *Fortschritte auf d. Geb. der Röntgenstrahlen*, 1905, VIII, 275  
 LUBOSCH, B. W. E., *Paris Medical*, 1929, I, 117  
 MACINTYRE, J., *Arch. Skag.*, 1897, I, 37  
 PORCHER (quoted by Van de Maele—see below)  
 RYFYNOLDS, R. J., Thesis, Cambridge University, 1925 (unpublished), *Proceedings of the Royal Society of Medicine*, 1934, XXVII, 985, *British Journal of Radiology*, 1934, VII, 415, *Ibid.*, 1935, VIII, 135, *American Journal of Radiology*, 1935, XXXIII, 522  
 ROUX, J., and BALTHAZARD, *Arch. Phys. Norm. et Path.*, 1898, X, 85  
 SABAT, 1911 (see article in *British Journal of Radiology*, 1934, VII, 705)  
 STUMPF, P., *Ibid.*, 1934, VII, 707  
 VAN DE MAELE, *Journal Belge de Radiologie*, 1935, XXIV, 265

- Arteriography in arteriovenous aneurysm, 157  
in differential diagnosis of chromophobe adenoma, 131  
in meningiomas, 140
- Arthritis, chronic, 458  
classification of B.M.A. committee, 459  
infective, of temporo-mandibular joint, 773  
non suppurative, acute, 456  
polyarticular, in children, diagnosis from osteochondritis of femoral head epiphysis 484  
rheumatic, acute, 456  
rheumatoid, primary, clinical features of, 460  
radiographic appearances of, 462  
secondary, clinical features of, 461  
suppurative acute, radiographic appearances of, 457  
tuberculous diagnosis from osteochondritis of femoral head epiphysis 483  
villous chronic 463
- Arthrodesis, combined intra and extra articular, 509  
extra articular 505  
intra articular, 504  
of knee, 508  
of shoulder, 511  
of tarsus 508
- Arthroplasty of hip, 507
- Astragalectomy Whitman's, 510
- Astragalo-saphenoid osseous of Pirie 279
- Astragalus, fracture-dislocation of, 383  
normal appearance of 274  
ossification of 277  
secondary, 280
- Astroblastoma, 143
- Astrocytoma, 143  
skull thinning in, 40
- Ateleiosis 347
- Atheroma of carotid or ophthalmic artery, 745
- Atlas abnormalities of 351  
fracture of, 388

## B

- Bamboo spine, 472
- Bankart's radical excision of hip joint 507
- Basal ganglia tumours of, ventriculography in, 106
- Basilar invagination of skull, 167
- Basophilism, 129, 554
- Bec de seau, 425
- Belot's projection in eye examination, 745
- Bismuth injections, opacities in soft tissues due to, 667
- Blastomycosis of bone 454
- Block vertebra, 499
- Bloodgood's classification of bone cysts, 637
- Blood vessel, cranial, changes in, in meningioma, 138
- Boeck's sarcoid diagnosis from gout, 537
- Bones, absorption of, hyperaemia in relation to, 337  
mechanism of, conclusions on 322  
under pathological conditions, calcium metabolism, 337

- Bones, absorption of under pathological conditions, effect of blood-supply on, 337  
effects of tumours on, 331  
general factors, 335  
inflammatory conditions, 329  
mechanical factors in, 324  
necrosis, 325
- actinomycosis of, 453
- and joints, deformities of, congenital, general considerations on, 341
- diseases of, of constitutional or unknown origin, 513  
effect of lesions of upper and lower motor neurons on, 495  
examination of, routine of, 251  
normal appearances of, general, 251  
in adults 252  
in children, 253  
static and paralytic lesions of, 493  
syphilis of, 436  
traumatic lesions of, 367  
late sequelae of, 389
- aneurysm of, 601
- blastomycosis of, 454
- blood supply of, effect of, on absorption and deposition 337
- calcium content of, changes in, 319
- cancellous, 305
- cement lines and "mosaic" structure of, 305
- cement substance in, 303, 304
- condylar granuloma of, 454
- compact, 305  
islands of, in adults, appearances of 243
- cysts of, 636  
arthritic, radiological appearances of, 641  
classification of Bloodgood's 637  
Knaggs's, 637  
radiological (Roberts), 638  
fibromatous, 642  
hydatid, radiological appearances of, 641  
in generalised diseases, radiological appearances of, 639  
myxochondromatous, radiological appearances of, 640  
simple, and fibrocytic diseases, relation between, 636  
and osteoclastoma relation between, 636  
radiological appearances of, 638  
traumatic, 333  
radiological appearances of, 640
- dead, reaction of tissues to, 325
- de-differentiation of into connective tissue  
histological evidence against, 318
- deposition of, 305  
mechanisms of, conclusions on 322  
under pathological conditions calcium metabolism, 337  
effect of blood supply on, 333  
effects of tumours on, 331  
general factors, 335  
inflammatory conditions, 329  
mechanical factors in, 324  
necrosis, 325
- diseases of, inflammatory, uncommon, 453

## C

- Calcaneo astragaloid, anterior, and astragaloid navicular joint space, 280  
 posterior, joint space, 280  
 Calcaneo cuboid joint space, 280  
 Calcaneum, epiphysis of osteochondritis of, 489  
 Calcaneus accessorius, 279  
   secundarius, 279  
 Calcification, cutaneous and subcutaneous, 654  
   due to therapeutic injections, 667  
   in arteries, 647  
   in hæmangioma of muscles, 648  
   in hæmatoma, 649  
   in soft tissues, 644-646  
   in veins, 647  
   of parasites, 650  
 Calcinosi circumscripta, 654  
   universalis, 655  
 Calcium balance, normal, 337  
   gluconate, injection of, opacities in soft tissues due to, 667  
   metabolism in relation to skeletal changes, 337  
 Calculus of brain, 64  
 Camp and Gianturco apparatus for radiography of optic foramen, 17  
 Canal, bony semicircular, anatomy of, 232  
 Capitellar epiphysis, ossification of, 265  
 Carcinomatosis differential diagnosis of, 567  
   osteoclastic, 565  
     radiological appearances of, 566  
   osteoplastic, 565  
     radiological appearances of, 567  
 Caries sicca, 425  
 Carpal bones, fractures of, 375  
 Carpo metacarpal joint space, 260  
 Carpus, normal appearances of, 256  
   ossification of, 262  
 Carte géographique, 538  
 Cauda equina, tumours of, 190  
 Caudate nucleus, tumours of, ventriculography in, 106  
 Cephalocoele, 166  
 Cerebellar tumours, ventriculography in, 110  
 Cerebrospinal fluid pathways, anatomy of, 72  
 Cervical rib, 352  
 Charcot's joint, 446  
   diagnosis from tuberculosis of knee, 433  
   radiological features of, 448  
 Chloroma, bone involvement in, 632  
 Cholesteatoma, complicating mastoiditis, 245  
   intracranial, 148  
   skull thinning in, 40  
   of skull, 177  
 Chondrodysplasia deforming, hereditary, 593  
 Chondroma, multiple, 517  
   diagnosis from gout, 537  
   radiological appearances of, 598  
   sites and pathology of, 597  
   sarcomatous changes in, 624  
   single, sites and radiological appearances of, 595  
 Chondro-osteodystrophy clinical and radiological features of, 516  
 Chondro-osteodystrophy, effect on intervertebral discs, 500  
   Hurler type, 517  
 Chordoma, intracranial, 150  
   of skull, 177  
 Choroid plexus, calcification of, 62  
 Chromophobe adenoma, 131  
 Cimeradiography, 763  
   apparatus for, Russell Reynolds, 767  
   clinical applications of, examination of alimentary tract, 778  
     of deglutition, 778  
     of effect of delay in emptying on normal stomach, 780  
     of filling of stomach, 779  
     of heart, 770  
     of joints, 780  
     of lungs, 776  
     of movements of normal stomach, 779  
     of thorax, 775  
   future of, 773  
   history of, 763  
   methods of, direct, 763-765  
     direct and indirect, comparison of, 772  
     Russell Reynolds apparatus and technique, 767-772  
     indirect, 763-767  
     indirect and direct, comparison of, 772  
     synthetic, 763, 765  
   technique of, Russell Reynolds, 767  
 Clavicle, examination of, 270  
   hæmangioma of, 604  
   inner end of, 270  
   ossification of, 270  
   outer end of, 270  
   shaft of, fracture of in adults, 370  
     in infants, 369  
 Cleido cranial dysostosis, hereditary, 349, 357  
 Clivoid processes, effect of intracranial tumours on, 49  
   normal, 46  
 Club hand, congenital, 358  
 Clutton's joints, 445  
 Coccygeal sinus, 669  
 Coccyx, examination of, 299  
 Cochlea, anatomy of, 232  
 Coeliac disease, skull in, 172  
 Colles' fracture, 373  
 Comberg's method of localisation of foreign bodies in eye, 757  
 Condyle of mandible, hypertrophy of, 740  
 Contrast media, use of, in diagnosis of breast tumours, 667  
   of cerebral conditions, 71, 88  
   of eye conditions, 745, 747, 758  
   of fistula and sinuses, 667  
 Cooley's anaemia, bone changes in, 559  
   skull changes in, 173  
 Cornea, examination of, 745  
 Corpus callosum, tumours of, ventriculography in, 106  
 Coxa vara, adolescent, 389  
   diagnosis from tuberculosis, 435 *See also* Epiphysis, slipped

- Epiphyseal scar, 253  
 Epiphyses, accessory, in children, 254  
   femoral, osteochondritis of, diagnosis from  
     tuberculosis of hip, 435  
   involvement of, in osteomyelitis, 409  
   of bones of foot, 276  
   of elbow joint, ossification of, 265  
   of shoulder joint, 269, 270  
   ossific nucleus of, in children, 253  
   slipped, aetiology of, theories of, 389  
   differential diagnosis of, 391  
   incidence of, 389  
   pathology of, 391  
   radiographic appearances of, 391  
 Epiphysitis, vertebral, 477  
 Ethmoid, osteoma of, 224  
 Ethmoidal cells, anatomy of, 204  
   developmental abnormalities of, 204  
   osteoporosis of, 224  
 Ewing's classification of sarcoma, 615  
   tumour and sclerosing osteomyelitis, simi-  
     larity of X ray appearances of, 409  
   clinical course of, 626  
   pathology of, 627  
   radiological appearances of, 628  
 Excision, radical (Bankart's), of hip joint,  
   507  
   simple, of hip joint, 507  
 Exophthalmos, unilateral, causes of, 58  
   examination of orbit and optic canal in, 58  
 Exostosis, apical, radiological appearances of,  
   708  
   excision of, 511  
   multiple, 593  
   nomenclature of, 587  
   single, 591  
 Extremity, lower, bones and joints of, normal,  
   271  
   malformation of, congenital, 361  
   upper, bones and joints of, normal, 255  
   malformations of, congenital, 356  
 Eye ear line, 9  
 Eye, foreign bodies in, 746 *See also* Foreign  
   bodies in eye  
   radiological examination of, 745

## F

- Fabella, examination of, 285  
 Facial bones, fractures of, 396  
 Fairbank's shelf operation, 507  
 Falx cerebri, calcified plaque of, 5, 6  
 Femur, abnormalities of, congenital, 361  
   head of, epiphysis of, ossification of, 290  
     osteochondritis of, 481  
   translucencies in, 289  
   lower end of, examination of, 284  
     fractures of, 380  
     ossification of, 287  
   neck of, fractures of, 377  
     base, 378  
     intertrochanteric, 378  
     midcervical, 378  
     subcapital, 377  
     union in, 378

- Femur, neck of, fractures of, osteochondritis  
   of, 489  
   plastered, 288  
   platymeric, 288  
   shaft of, examination of, 288  
     fractures of, 378  
   upper end of, examination of, 288  
     osteomyelitis of, diagnosis of, from  
       tuberculosis of hip, 435  
     structure of, 289  
 Fibrocystic disease and simple cyst of bone,  
   relation between, 636  
   focal, diagnosis from xanthomatosis, 540  
 Fibrosarcoma, medullary and periosteal, 623  
 Fibula, coultured, 282  
   head of, ossification of, 286  
   lower end of, examination of, standard  
     views for, 281  
     ossification of, 282  
   malformations of, congenital, 361  
   shaft of, examination of, 282  
   upper end of, examination of, 283  
     fracture of, 381  
 Filaria Bancrofti, calcification of, 653  
   medinensis calcification of, 654  
 Fistula, branchial, 669  
   complicating mastoiditis, 247  
   preauricular, 669  
   thyroglossal, 668  
   umbilical urinary, 669  
 Flat foot, 495  
 Fluoroscopy for localisation of foreign bodies,  
   673, 675  
 Foot, accessory ossicles of, 277  
   bones of, ossification of, 276  
   examination of, standard views, 271  
   flat, 495  
   malformations of, congenital, 361  
 Foreign bodies in eye, detection of, 746  
   localisation of, general points in, 747  
   methods of, Ahlborn's, 756  
     Arganaraz's, 756  
     bone free, 757  
     Comberg's, 757  
     contrast medium, 758  
     Franceschetti's, 758  
     geometric, 748  
     Henrard's, 756  
     Lindblom's, 758  
     McGrigor's, 753  
     physiological, 748  
     simple, 756  
     stereoscopic, 756  
     Sweet's, 748  
     Velter's, 757  
     Vogt's, 757  
     Wessely's, 757  
   types of, 746  
   in jaw, 707  
   in orbit, 746  
   localisation of, 672  
     methods of direct observation, fluoro-  
       scopy, 673  
     parallax principle, 673  
     stereoscopic, 675

- Foreign bodies, localisation of, methods of,  
 similar triangle, double-exposure film  
 method, 676  
     double film method, 676  
     fluoroscopic, 675  
     McGrigor's, 675  
     Mackenzie Davidson's, 677
- Fractures, compound, infected, osteomyelitis  
 in, 413  
     diagnosis of, mistakes in, 367  
     recognition of, 367  
     union of, 368  
*See also under names of bones*
- Fraenkel, white line of in scurvy 531
- Fragilitas osseum of skull, 167
- Franceschetti's method of localisation of  
 foreign bodies in eye 758
- Frankfurter horizontal plane, 9
- Freiberg's infraction, 485
- Frontal lobe tumours, angiography in 122
- G
- Gangrene, gas, 662  
     radiological appearance of, 663
- Gargoylism, 517
- Garré's sclerosing osteomyelitis, 409
- Gas gangrene, 662  
     diffuse, radiological appearance of 663  
     localised, radiological appearance of, 663
- Gaucher's disease, 540
- Geo's disease, 528
- Genu valgum, 494  
     varum, 494
- Gerüstmarkzone of Lehnendorff in scurvy, 531
- Gigantism, acromegalic, 129  
     clinical and radiological features, 552  
     skull changes in, 170
- Girdlestone's operation for hallux valgus, 511
- Glenoid cavity, fracture of, 371
- Globlastoma multiforme, 143
- Gliomas, 141  
     calcification in, 65  
     radiological signs of, 143  
     skull thinning in, 40
- Globe, shrunken calcified, appearance of 745
- Gout, clinical features of, 535  
     differential diagnosis of, 537  
     radiological appearances of, 536
- Graham Hodgson sinus stand, 210  
     upright radiographic stand, 210
- Granuloma apical 695  
     coccioid, of bone, 454
- Granulomatosis, lipid, 537
- Guinea worm, calcification of, 654
- Gummata of bone radiographic appearances  
 of, 444
- H
- Hæmangioma, cavernous of skull, 38, 176  
     of muscles, calcification in, 648  
     of vertebrae, 189
- Hæmatoma, calcification in 649  
     subdural, 158  
     skull thinning in, 40
- Hæmophila, joint changes in, 572
- Hæmorrhage, intracranial, 174
- Habsteresis, 319  
     evidence against, 321
- Hallux valgus, operations for, excision of base  
 of proximal phalanx, 511  
     excision of exostosis, 511  
     excision of metatarsal head, 511  
     Girdlestone's, 511
- Hand and wrist, examination of, standard  
 positions, 235  
     ossification of, 261  
     deformities of, congenital 358  
     interphalangeal joints of, osteo arthritis of  
     466  
     lobster claw deformity of, 359, 360  
     rheumatoid arthritis of, 463
- Haversian space, 307, 308  
     systems, 303, 307, 310
- Head, boat shaped, 167  
     injury, penetrating, effects of, 112  
     oblique or flattened, 167  
     steeply, 166
- Heart, examination of, by cinematography, 775
- Hemieranosis, 172
- Hemmelus, 356
- Henrad's method of localisation of foreign  
 bodies in eye, 756
- Hip joint, dislocation of, 379  
     congenital, 362, 380  
     examination of, in abducted latero  
     medial and medio lateral views, 290  
     in abduction, 290  
     in external rotation, 290  
     in normal position, 288  
     standard positions for, 288  
     operations on, arthrodesis, varieties of, 504  
     arthroplasty, 507  
     excision, 507  
     insertion of Smith Petersen pin, 509  
     osteotomy, varieties of, 504  
     shelf (Fairbank), 507  
     ossification in region of, 290  
     osteo-arthritis of, 466  
     osteochondritis of, diagnosis from slipped  
     epiphysis, 392  
     tuberculosis of, 433  
     diagnosis from slipped epiphysis, 391  
     differential diagnosis of, 435
- Hodgkin's disease, 562  
     bone involvement in, 532  
     radiological appearance of, 563  
     skull thinning in, 40
- Hot cross bun skull 439
- Howship's lacunæ, 305, 311, 312
- Humerus, head of, examination of, 267  
     lower part of, displacement of epiphyses of  
     epicondyles, 372  
     examination of, 264  
     fractures of, extension, 372  
     flexion, 372  
     supracondylar, 372  
     malformations of, congenital, 367  
     shaft of, examination of, 266  
     upper part of, displacement of epiphyses  
     of, 371

- Humerus, upper part of, fractures of, 371  
 abduction, 371  
 adduction, 371  
 anatomical neck, 371  
 contusion crack, 371  
 greater tuberosity, 371  
 ossification of, 269  
 Hurler type of chondro osteodystrophy, 517  
 Hutchinson type of suprarenal neoplasm, 555  
 Hydatids of bone, 641  
 brain, 152  
 Hydrocephalus, adult, 161  
 infantile, 160  
 Hyperæmia in relation to bone absorption, 333  
 Hypercementosis, radiological appearances of, 708  
 Hyperostosis calvaria diffusa, 171  
 crani, 171  
 frontalis interna, 171  
 of meningioma, 37, 138  
 of skull, 25, 27  
 Hyperparathyroidism, 542  
 bone pathology of, 336  
 calcium balance in, 338, 339  
 skull changes in, 170  
 Hypertelorism, 347  
 Hyperthyroidism, bone pathology of, 336  
 Hypervitaminosis D, bone changes in, 336  
 Hypocalcification, 319  
 Hypodermolipomatosis, 654  
 Hypothyroidism, diagnosis from osteochondritis of femoral head epiphysis, 483

## I

- Icterus, hæmolytic, familial, 560  
 Ilium, hæmangioma of, 604  
 Infantile, 546  
 Lorain type of, 546  
 radiological appearances of bones in, 548  
 skull changes in, 170  
 Injections therapeutic, opacities in soft tissues due to, 667  
 Intercalary bones, 500  
 Interphalangeal joints of hands, osteoarthritis of, 466  
 Intervertebral discs, 294, 498  
 abnormalities of, 499  
 congenital, 499  
 absence of, 499  
 calcification of, 500  
 changes produced in by disease, 502  
 degenerative and traumatic lesions of, 500  
 effect of chondro osteodystrophy on, 500  
 effect of lumbar puncture on, 500  
 effect of primary congenital defect in spine on, 499  
 herniation of, 189, 190  
 into vertebral body, 501  
 into vertebral canal, 502  
 normal anatomy of, 498  
 annulus fibrosus of, 498  
 disc plates of, 498  
 nucleus pulposus of, 498  
 progressive change in, radiographic appearances of, 498

- Intervertebral discs, nuclear expansion of, 500  
 senile degeneration of, 500  
 Intracranial complication of mastoiditis, 246  
 examination, antero posterior projection, 11  
 axial projection, 15  
 cephalography, 71, 87  
 half axial postero anterior projection, 15  
 lateral projection, 9  
 occipito frontal projection, 15  
 planigraphy in, 21  
 postero anterior projection, 11  
 routine, 9  
 submento vertical projection, 15  
 technique of, 7  
 tomography in, 21  
 ventriculography, 71, 81  
 pathology in relation to radiology, 21  
 pressure, rise of radiological appearances of, 12  
 tumours, acoustic neuroma, 145  
 calcification in, 25, 65  
 cholesteatoma and allied tumours, 184  
 chordoma, 150  
 colloid cyst of third ventricle, 146  
 displacements due to, 24  
 effect on sella turcica, 49  
 giving normal ventriculograms, 89  
 glioma, 141  
 hydatids, 151  
 local effects of, on skull, 25  
 meningeal vessels in relation to, 44  
 meningioma, 135  
 metastatic, 37, 146  
 papilloma, 150  
 parasitic cysts, 151  
 pituitary, 129  
 radiological diagnosis of, faulty, 6  
 signs of, summary of, 127  
 reaction of skull to, 26  
 sella and suprasellar epidermoid, 132  
 symptoms of disorder of endocrine and vegetative functions, 128  
 early, 127  
 epilepsy, 128  
 focal, 128  
 hydrocephalic, 127  
 progressive dementia, 128  
 swelling, 128  
 thinning of skull in, 40  
 tuberculoma, 151  
 varieties of, 126  
 vascular changes in, 25  
 ventriculograms of, 89 *et seq*

## J

- Jaws, changes in, in acromegaly, 733  
 clicking, 738  
 cysts of, 719  
 diseases of, inflammatory, 733  
 foreign bodies in, 707  
 fractures of, 712  
 pathological, 735  
 necrosis of, 737  
 osteitis fibrosa of, 733  
 osteomyelitis of, 733  
 tumours of, 723

- Jaws, tumours of, benign giant celled** 727  
     *carcinoma, basal celled*, 729  
     secondary, 729  
     epithelioma, 729  
     myeloma, 729  
     odontomes, 723  
     osteoma, 732  
     sarcoma, 732
- Joints and bones, deformities of, congenital,**  
     general considerations on, 341  
     arthritis of, diagnosis from syphilis, 446  
     capsule in children, 254  
     Charcot's, 446  
     Clutton's, 445  
     examination of, by cinematography, 780  
     routine of, 251  
     involvement of, in osteomyelitis, 410  
     normal appearances of, general, 251  
         in adults, 252  
         in children, 253  
     syphilis of, acquired, 446  
         differential diagnosis of, 446  
         congenital, 445  
     tuberculosis of, incidence of, 423  
         mode of infection in, 423  
         pathology of, 423  
         radiological appearances of, stage of  
             invasion, 424  
             stage of progress, 424  
             stage of repair, 425  
     *See also under name of joint*
- Jüngling's osteitis tuberculo-sa multiplex cysto-**  
     *ides*, 415
- K
- Kalksicht**, 654
- Kashin Beck's disease**, 557
- Kienbock's disease**, aseptic bone necrosis in  
     relation to, 326  
     diagnosis from crush fracture, 376
- Klippel Feil syndrome**, 351
- Knaggs's classification of bone cysts**, 637
- Knee joint, arthritis of, diagnosis from tuber-**  
     *culosis*, 433  
     dislocation of, 381  
         congenital, 365  
     examination of, standard positions for,  
         283, 284  
     fractures in region of, 380  
     operations on, arthrodesis, 504  
     excision of patella, 508  
     supracondylar osteotomy, 508  
     ossification in region of, 286  
     soft tissues around, 286  
     space, 286  
     syphilis of, diagnosis from tuberculosis, 431  
     tuberculosis of, 432  
         differential diagnosis of, 437
- Köhler's disease**, aseptic bone necrosis in  
     relation to, 326  
     clinical features and radiographic appear-  
     ances of, 484, 485, 487
- Kummell's disease**, 388, 393
- Kyphosis dorsalis adolescentum**, 477  
     *juvenilis*, 477

- Kyphosis, juvenile**, 477  
     *osteochondropathic*, 477

## L

- Labyrinth, anatomy of**, 232  
     ethmoidal, examination of, 204  
     examination of, technique of, 234
- Lachrymal glands, radiological appearances**  
     of, 768
- Lambert's operation for drop foot**, 510
- Lamina dura, normal**, 683, 689
- Lead poisoning, bone changes, in**, 556  
     diagnosis from osteopetrosis, 570  
     diagnosis from scurvy, 434
- Lehndorff, geristmarkzone of, in scurvy**, 531
- Lens, calcification of**, 745
- Leontiasis ossea**, 568  
     of skull, 38
- Leprosy, bone changes in**, 452, 497  
     differential diagnosis of, 452  
     diagnosis from syphilitic dactylitis, 440
- Leukæmia, bone deposits in**, 562
- Lymphatic, skull thinning in**, 40
- Lungs, lower, malformations of, congenital**,  
     351  
     osteochondritis of, primary and secondary  
     centres of, 474  
     upper, malformations of, congenital, 356  
     osteochondritis of, primary and secondary  
     centres of, 474
- Lundblom's method of localisation of foreign**  
     bodies in eye, 758
- Lines of arrested growth in bones**, 253
- Lipiodol, external injection of**, 193  
     in diagnosis of fistula and sinuses, 668  
     of lachrymal gland lesions, 759  
     of nasal sinus disease, technique of, 226  
     of spinal cord tumours, 191  
     intrathecal injection of, 191  
     lumbar injection of, 194
- Lipoma of bone**, 633  
     radiological appearance of, 665
- Little's disease, skull abnormalities in**, 169
- Localiser, McGrigor's**, 675  
     MacKenzie Davidson's, 677  
     Sherton's, 674
- Looser's zones**, 528
- Lorain type of infantile**, 546
- Lorenz bifurcation osteotomy**, 504
- Luckenschädel**, 346
- Lumbar puncture, effect of on intervertebral**  
     discs, 500
- Lumpy jaw of cattle**, 454
- Lungs, examination of by cinematography**, 776
- Lymphogranuloma**, 662  
     radiological appearances of bone involve-  
     ment in, 567
- Lymphosarcoma, skull thinning in**, 40
- Lynch table, use of, in intracranial examina-**  
     tion, 8
- M
- McGrigor's fixed screen method for localisation**  
     of foreign bodies, 675  
     *localiser*, 675



- McGrigor's method for localisation of foreign bodies in eye, 753  
moving screen method for localisation of foreign bodies, 676
- Mackenzie Davidson's localiser, 677
- McMurray's osteotomy, 504
- Madura foot, 455
- Malacia of vertebra, effect on intervertebral discs, 503  
post traumatic, 475
- Malar bones, fractures of, 398
- Malgaigne's fracture, 385
- Malta fever, bone lesions in, 452
- Mammography, contrast, 667
- Mandible, carcinomas of, 729  
condyle of, hypertrophy of, 740  
dental cysts of, 719  
dentigerous cysts of, 721  
fractures of, 400, 712  
displacement in, amount of, 713  
in children, 716  
number and sites of, 713  
pathological, 715  
progress of treatment, 715  
relation of teeth to line of, 714  
type of, 713  
normal, coronoid and condylar processes, 685  
genio hyoid tubercle, 685  
inferior border, 684, 688  
inferior dental canal, 684  
internal and external oblique ridges, 685  
mental foramen, 685  
osteitis fibrosa of, 733  
osteomyelitis of, 411, 733  
periostitis of, 737  
unerupted supernumerary teeth in, 692
- Marble bones, 534, 568  
skull changes in, 174
- March fracture, 384
- Mastoid process, anatomy of, 232  
diseases of, 237  
pneumatisation of, 232  
post operative, 238, 247  
radiological examination of, interpretation of, 243  
lateral oblique position, 241  
postero anterior oblique position, 241  
scope of, anatomical, 239  
pathological, 240  
30° fronto occipital view, 241
- Mastoidectomy, cortical, 238  
radical 239
- Mastoiditis, acute, etiology and pathology of, 237  
in cellular mastoid 243  
chronic, 238  
in cellular mastoid, 244  
complications of, 245  
in acellular mastoid, 244  
zygomatic, 247
- Maxilla, carcinoma of, 729  
dental cysts of, 719  
dentigerous cysts of, 721  
fractures of, 716  
normal, anterior palatine foramen, 686  
coronoid process, 688
- Maxilla, normal, hamular process, 688  
incisive fossae, 686  
inter maxillary suture, 686  
malar bone, 687  
maxillary antrum, 687  
nasal fossae, 686  
nasal septum, 686  
osteitis fibrosa of, 733  
unerupted supernumerary teeth in, 690
- Meatus, internal auditory, examination of, 60
- Medulloblastoma, 143
- Melorheostosis (*Leri*), 571
- Meningiomas, 135  
arteriographic appearances, 140  
calcification in, 66, 140  
cerebello pontine angle, 138  
differential diagnosis of, 141  
fronto temporal, radiological appearance of, 4  
hyperostosis of, 37  
olfactory groove, 137  
parasagittal, 136  
radiological signs of, 138  
skull thinning in 40  
sphenoidal ridge, 137  
suprasellar, 136
- Meningocele, 166, 354
- Meningomyelocele, 354
- Mercury injections, opacities in soft tissues, due to, 667
- Metacarpals, bases of, normal appearances of, 256  
epiphyses of, abnormal, 261  
head of, normal appearance of, 255
- Metacarpus, normal appearances of, 255  
ossification of, 261
- Metastases, clinical features of, 605  
intracranial, 146  
osteoblastic, 611  
osteolytic, 608  
radiological appearances of, 607  
sclerosing, 611
- Metatarsal, first, normal appearance of, 272  
four outer, normal appearance of, 273  
fractures of, 384  
head of, excision of, 511  
osteochondritis of, 485
- Metatarsus, examination of, 272  
ossification of, 276
- Microcephaly, 166  
vera, 166
- Micromelia, 511
- Midbrain, tumours of, ventriculography in, 107
- Mid carpal joint space, 260
- Mongolism, clinical features of, 549  
radiological appearance of bones in, 550  
skull changes in, 170
- Morbus coxae senilis, 466
- Morgagni's syndrome, 171
- Morquio Brailsford's dyschondroplasia diagnosis from osteochondritis of femoral head epiphysis, 484
- Motor neurone, lower, lesions of, bone and joint changes in, 495  
upper, lesions of, bone and joint changes in, 495

- Mucocele of ethmoid cell or frontal sinus, 225  
 Mycetoma pedis, 435  
 Myelocoele, 354  
 Myelographic examination of spinal cord, 197  
 Myeloma, clinical features and pathology of, 630  
 endothelial, 626  
 of skull, 177  
 osteogenetic, of vertebrae, 188  
 plasma celled, spinal compression in, 189  
 radiological appearances of, 632  
 Myelomatosis, diagnosis from xanthomatosis, 539, 540  
 multiple, 567, 630  
 diagnosis from carcinomatosis, 567  
 from generalised osteitis fibrosa, 543  
 of jaws, 729  
 spinal compression in, 189  
 Myosarcoma of bone, 633  
 Myositis ossificans progressiva, 659  
 traumatic, 589  
 radiological appearances of, 658  
 Myxodema, infantile, bone changes in, diagnosis from osteochondritis of femoral head epiphysis, 483

## N

- Nasal bones, fractures of, 397  
 Naughton Dunn's operation, 509  
 Naviculo-cuneiform joint space, 260  
 Necrosis, peri-apical, 198  
 Nerve, eighth, tumour of, 238  
 Nervous system, central, radiographic examination, general technique and pathology, 3  
 Neurofibroma, acoustic, 145  
 Neurofibroma of eighth nerve, 236  
 spinal compression in, 189  
 Niemann Pick's disease, 541  
 Nose, fractures of, 397  
 Nucleus pulposus, 294

## O

- Odontome, composite, complex, 721  
 compound, cystic, 723  
 non cystic, 723  
 dilated, 725  
 epithelial, 727  
 follicular, 721  
 geminated, 724  
 Olecranon epiphysis, ossification of, 265  
 process, fracture of, 374  
 Oligodendroglioma, 145  
 Ollier's disease, pathology and radiological appearances of, 599  
 Operations, orthopaedic, in relation to radiography, 501  
 Optic canal, normal and pathological appearances of, 59  
 foramen, examination of, projection for, 17  
 Orbit, foreign bodies in, 706  
 upper half of, examination of, projections for, 19  
 Orbito mental line, 9  
 Orthopaedic operations in relation to radiography, 501  
 Os acromiale, examination of, 268

- Os calcis, fractures of, 393  
 lamellar struts of, 276  
 normal appearance of, 273  
 osteomyelitis of, 413  
 Os capitatum, normal appearance of, 256  
 Os centrale, 259  
 Os hamatum, normal appearance of, 256  
 Os intercuneiforme, 279  
 Os intermetatarsale, 280  
 Os lunatum, normal appearance of, 257  
 Os magnum, normal appearance of, 256  
 Os multangulum majus, normal appearance of, 256  
 Os multangulum minus, normal appearance of, 256  
 Os naviculare, normal appearance of, 256  
 Os paracuneiforme, 280  
 Os styloideum, 260  
 Os subtibiale, 260  
 Os sustentaculum proprium, 278  
 Os tibiale externum, 279  
 Os trapezoides secundarium, 260  
 Os triangulare, 260  
 Os trigonum, 277  
 Os triquetrum, normal appearance of, 257  
 Os ulnare externum, 260  
 Os vesibulum, 260, 260  
 Osgood's disease, 488  
 Ossiculum trochlea, 279  
 Ossification in soft tissues, 655  
 in tendons, 656  
 Osteitis apical radiating, acute, 698  
 chronic, 700  
 in long bone, 702  
 deformans 573  
 amorphous form 574  
 bones affected in, order of, 574  
 diagnosis from generalised osteitis fibrosa 543  
 from xanthomatosis 540  
 of skull, 78  
 osteogenic sarcoma in, 579  
 pathology of, 335  
 radiological appearances of arteries, 576  
 of bones of extremities 577  
 of pelvis, 574  
 of skull 577  
 of spine, 576  
 sarcomatous changes in, 624  
 skull changes in, 174  
 spongy form, 574  
 fibrous, focal, 571  
 skull changes in, 173  
 generalised, clinical features of, 542  
 differential diagnosis of, 543  
 radiological appearances of, 542  
 skull changes in, 170  
 of jaws, 733  
 of skull, 38  
 gummatous, 445  
 multiplex cystica, leprosy, 452  
 pyogenic, of skull, 174  
 sclerosing, of wall of left antrum, 224  
 syphilitic, acquired, diffuse, radiographic appearances of, 443

- Osteitis, syphilitic, acquired, localised, radiographic appearances of, 444  
late congenital, radiographic appearances of, 441  
of skull, 175
- Osteitis tuberculosa multiplex cystoides (Jungling), 415  
diagnosis from gout 537  
radiographic appearances of, 416  
tuberculous of skull, 175
- Osteo-arthritis, diagnosis from syphilis, 446  
of temporo mandibular joint, 738  
radiographic appearances, 464  
clinical significance of, 469
- Osteo arthropathy, hypertrophic, clinical features of, 557  
radiological appearances of, 558
- Osteoblasts, 304
- Osteochondritis, 474  
adult, 475  
aetiology of, 475  
clinical features of 476  
coxæ juvenilis 481  
deformans, 481  
diagnosis from tuberculosis of shoulder, 430  
dissecans, aseptic bone necrosis in relation to, 326  
clinical features and radiological appearances of, 490  
juvenile, 475  
morbid anatomy of, 476  
of calcaneal epiphysis, 489  
of femoral head epiphysis diagnosis from tuberculosis of hip, 435  
clinical and radiological phases, table of, 482  
clinical features of, 481  
differential diagnosis of, 483  
radiographic appearances of, 482  
of femoral neck, 489  
of metatarsal heads, clinical features of, 485  
radiographic appearances of, 487  
of patella 489  
of tarsal scaphoid, clinical features and radiographic appearances of, 484  
of tibial tubercle clinical features and radiographic appearances of, 488  
of vertebral epiphyseal plates clinical features of, 477  
radiographic appearances of, 478  
synonyms of 477  
primary and secondary centres of, in spine and extremities, 474  
radiological observations in, 477  
syphilitic 436  
radiographic appearances of 437  
vertebral 477
- Osteochondroma, 590 591  
multiple, 593  
clinical features and pathology of, 594  
radiological appearances of, 595  
sarcomatous changes in, 624  
single, clinical features and pathology of, 592  
radiological appearances of, 593
- Osteoclasia in inflammatory conditions, 329
- Osteoclastoma and simple cyst, relation between, 636  
clinical features of, 600  
of jaw, 727  
pathology and radiological appearances of, 601
- Osteoclasts, bone-eroding powers of, 313  
characters of, 311  
fate of, 313
- Osteogenesis imperfecta, clinical features of, 215  
of skull, 167  
radiological appearances of, 520
- Osteolysis 318, 323
- Osteoma intracranial, 67, 588  
of skull 38, 176  
pathology of, 587  
simple, radiological appearances of, 588  
traumatic, pathology of, 589  
radiological appearances of, 590, 658
- Osteomalacia and rickets, radiological difference in, 522  
bone changes in, 319  
calcium balance in, 338, 339  
diagnosis from generalised osteitis fibrosa 543  
dietetic, 524  
due to idiopathic steatorrhœa, 528  
radiological appearances in, 526  
skull changes in, 172
- Osteomyelitis, complications and sequelæ of, 409  
definition of 401  
diagnosis from tuberculosis of hip joint, 435  
fibrous, chronic 642  
hæmatogeneous acute, pathological features, 402  
radiological features of, 403  
of jaws, 733  
sclerosing (Garre), 409  
special sites of, 411  
subacute and chronic, 406  
syphilitic, early congenital, radiographic appearances of, 438  
late congenital radiographic appearances of, 441
- Osteopetrosis 568  
differential diagnosis of, 570  
radiological appearances of 569
- Osteopokaliæ, 599
- Osteopokulosis 517
- Osteoporosis, 319  
circumscribed, skull changes in, 174  
of ethmoid cells, 224  
of skull, 26  
thyrotoxic, 544
- Osteopsathyrosis idiopathica 518
- Osteosclerosis fragilis generalisata, 568
- Osteosclerosis of skull, 25
- Osteotomy, intertrochanteric, 504  
Lorenz bifurcation, 504  
McMurray's, 504  
subtrochanteric, 504  
supracondylar, 508  
wedge, 504
- Otosclerosis, radiological appearance of, 23

Oxycephaly, 166, 344

Oxygen as contrast media in ventriculography and encephalography, 88

## P

Paget's disease of bone, mosaic structure in, 310

pathology of, 335

sarcomatous changes in, 624

See also Osteitis deformans

Papilloma intracranial, 159

Paradental disease, pathology and radiographic appearances of, 695

Parallax principle in localisation of foreign bodies, 673

Paravertebral tumours causing spinal compression, 189

Parietal lobe tumours, angiography in, 123

Patella, absence of, congenital, 366

bipartita, 285

cutiti, 358

dislocation of, 381

congenital, 366

emargination of, 285

examination of, 285

excision of, 508

fractures of, 381

multipartita, 285

ossification of, 287

osteochondritis of, 489

Pelkan, lateral spur of, in scurvy, 533

Pellegrini Stieda disease, 392, 610

Pelvis, abnormalities of, congenital 355

examination of, 301

fractures of, 384

Negele, 355

osteomyelitis of, 412

Robert, 355

Pepper type of supratentorial neoplasm, 553

Periodontal disease, pathology of, 695

radiographic appearances of, 695

membrane, inflammatory diseases of, 695

normal, 681

Periodontitis, productive, radiological appearances of, 708

rarefying radiological appearances of, 709

Periostitis, 401

syphilitic, acquired radiographic appearances of, 442

early congenital, radiographic appearances of, 438

late congenital, radiographic appearances of, 440

Perthes' disease, aseptic bone necrosis in relation to, 326

Perthes-Legg's-Calve's disease, 481

Petrositis, apical, complicating mastoiditis, 247

Petrous bone, anatomy of, 230

examination of, 60, 235

technique of, 234

vertical projection, 18

fractures of, 236

tumours of, neurofibroma, 236

sarcoma, 236

Phalanges of foot, distal, 271

mid 271

ossification of, 276

proximal, 272

excision of base of, 511

of hand, mid, 255

normal appearances of, 265

proximal, 255

ungual, 255

of thumb, 255

ossification of, 261

osteomyelitis of, 411

Phlebotomy, 647

Phocomelia 356

Phosphorus poisoning, bone changes in, diagnosis from scurvy, 424

Pineal body, calcification of, 61

Pink spot, 709

Pisiform normal appearance of, 257

secundarium 260

Piso cuneiform joint space, 260

Pituitary adenomas, varieties of, 129

tumours, 129

angiography in, 123

sella enlargement in, 51

Plagiocephaly, 167, 346

Planigraphy of skull, 21

Platycnemid tibia, 282

Polydactyly 359, 361

Polyp of antrum, 223

Pontine tumours, ventriculography in, 111

Forus acusticus, examination of, lateral projection 18

vertical projection 18

Posture defects of 493

Pott's disease, 419

fracture, 382

Precocious clinical and radiological features of, 552

Proetz displacement method, technique of, 226

value of, 227

Projections for intracranial examination, 9

Pseudo coxalgia, 48

fractures in osteomalacia 527

-oxycephaly, 345

Psoriasis arthropathica, 578

Pulp nodules or stones in teeth, 707

Pvorrhica, pathology and radiographic appearances of 69

## Q

Queckenstedt's test, use of, 192

## R

Radio-carpal joint space, 260

Radio ulnar, inferior, joint space, 260

Radius and ulnar shafts, examination of, lateral supinated view, 262

lateral view, 263

paino-dorsal view, 262

Incipital tuberosity of, epiphysis of, ossification of, 265

- Radius, lower end of, fractures of, 373  
     Colles, 373  
     normal appearance of, 258  
     ossification of, 262  
     malformations of, congenital, 357  
     upper end of, examination of, 263  
     fractures of, neck, 373  
     vertical crack in head, 373
- Rathke's pouch, tumour of, 132
- Reid's baseline, 9
- Reynolds, Russell apparatus and technique for cineradiography, 767
- Rheumatic diseases, effect on intervertebral discs, 502
- Rheumatoid arthritis, 460 *See also* Arthritis, rheumatoid
- Ribs, abnormalities of, 350, 352, 353  
     cervical, 352  
     fractures of, 385
- Rickets, adolescent 523  
     and osteomalacia radiological difference in, 522  
     bone changes in, 319  
     calciun balance in, 338, 339  
     clinical features of, 522  
     coeliac, 528  
     diagnosis from scurvy, 534  
     radiological appearances of, 522  
     renal, diagnosis from rickets due to vitamin D deficiency, 524  
     from xanthomatosis, 540  
     scurvy deformity in, 523  
     skull changes in, 172
- Rider's bone, 590, 659
- Ring localiser, 673
- Roberts radiological classification of bone cysts, 638
- Russell Reynolds apparatus and technique for cineradiography, 767-772
- 5
- Sabre tibia, 441, 452
- Sacralisation of fifth lumbar vertebra, 353
- Sacro iliac joint, tuberculosis of, 430  
     differential diagnosis of, 431
- Sacrum, examination of, 299
- Sarcoma, capsular and parosteal, 624  
     neurogenic, of bone, 633  
     of skull, 177  
     of spine 189  
     osteogenic, 611  
     capsular and parosteal, 624  
     classification of, 615  
     American College of Surgeons, 615  
     Ewing's, 615  
     clinical features of, 613  
     extraperiosteal, 621  
     fibrosarcoma, 623  
     pathology of, 614  
     periosteal, 621  
     radiological appearances of, 615  
     bone destruction, 617  
     effect of previous irradiation of, 626  
     mixed nature of, 619

- Sarcoma, osteogenic, radiological appearances of, soft tissue tumour, 620  
     subperiosteal new bone formation, 618  
     sclerosing, 620  
     superimposed on pre-existing disease, 624  
     telangiectatic, 620
- Scaphocephaly, 167, 346
- Scaphoid, carpal, bipartite, 376  
     double, 257  
     fractures of, 375  
     normal appearance of, 256  
     ossification of, 277  
     tarsal, fractures of, 384  
     normal appearance of, 273  
     osteochondritis of, 484
- Scapula, examination of, 267  
     fractures of, 370  
     haemangioma of, 604  
     ossification of, 269
- Scheuermann's disease, 477  
     radiographic appearances of, 478
- Schlatter's disease, 488  
     aseptic, bone necrosis in relation to, 326
- Schnorr's nodes, 478, 501
- Schuller's disease, clinical features, 629  
     radiological appearances of, 630
- Schuller Christian syndrome, 537  
     skull changes in, 172
- Scoliosis, 493
- Scurvy, clinical features of, 530  
     differential diagnosis of, 533  
     radiological appearances of bones in, 531
- Sella turcica, anatomical features of, 46  
     calcification in or near, causes of, 54  
     destruction of, causes of, 53  
     effect of intracranial pressure on, 23  
     enlargement of, pathological, 48  
     examination of, lateral projection for, 17  
     normal appearances of, 46  
     pathological appearances of, 48  
     smallness of, pathological, 48  
     thickening of, causes of, 54  
     variations in shape of, normal, 47  
     variations in size of, pathological, 48
- Sellar and suprasellar epidermoid tumour, 132
- Semilunar bones, fractures of, 376  
     normal appearance of, 257
- Septum lucidum, tumours of, ventriculography in, 106
- Sequestra, formation of, in inflammatory bone conditions, 329
- Sesamoid bones in first metatarso phalangeal joint, 272  
     in hand, normal appearance of, 257  
     in toes, 272  
     ossification of, 262
- Sesamum peroneum, 280
- Shenton's line, 290  
     localiser, 674
- Shoulder girdle, malformations of, congenital, 356  
     joint, arthritis of, diagnosis from tuberculosis, 429, 430  
     bones of, ossification of 269

- Shoulder joint, dislocation of, 372  
     congenital, 362  
     examination of, standard position, 266  
         variations in, 268  
     operations on, arthrodesis 511  
     osteo arthritis of, 466  
     space, 268  
     tuberculosis of, 427  
         differential diagnosis of, 429  
*Sinus stand, Graham Hodgson, 210*  
*Sinuses, cervical, 669*  
     coccygeal, 669  
     dural, normal appearances of, 42  
     frontal, anatomy of, 202  
         examination of, 202  
         osteoma of, 224  
     maxillary, anatomy of, 201  
         examination of, 201  
     nasal, accessory, anatomy of, 201  
         bone changes in, 224  
         carcinoma of, 225  
         cyst of, 222  
         dental cyst invading 225  
         diseases of, 217  
             diagnosis of by lipiodol, technique  
                 of, 226  
             by Proetz displacement method,  
                 226  
         examination of, importance of erect  
             posture, 209  
             importance of standardised tech  
                 nique, 209, 210  
             normal transradiancey of, 209  
             radiological interpretation of sinu  
                 sitis, 218  
             technique of, 207  
                 lateral view, 213  
                 left oblique view, 213  
                 occipito frontal view, 210  
                 occipito mental view, 211  
                 right oblique view, 213  
                 vertex mental view, 212  
     mucocoele of, 225  
     osteoma of, 224  
     polyp of, 223  
     stereoscopic examination of, importance  
         of, 206  
     tumours of, 224  
     pilonidal, 669  
     sphenoidal, anatomy of, 205  
         examination of, 203  
*Sinusitis, diagnosis of, by lipiodol, 226*  
     by Proetz displacement method, 226  
     hyperplastic, 217  
     radiological interpretation of, fluid levels, 219  
         gross opacity, 218  
         thickened lining membrane, 221  
     suppurative, 217  
*Skeleton, normal growth and reconstruction of,*  
     by bone erosion and deposition, 307  
*Skull, abnormalities of, 31*  
     due to endocrine and constitutional dis  
         eases, 169  
     acrocephalic, 345  
     anomalies and diseases of, 165  
*Skull, arterial grooves of, diagnosis from frac  
     tures, 369*  
     base of lesser wing of sphenoid, normal  
         appearances of, 55  
         pathological appearances of, 57  
         methods of examination, 55  
         pathological appearance of, 55  
     basilar invagination of, 167  
     birth injuries of, 183  
     cavernous haemangioma of, 38  
     cyst of, simple, 177  
     defects of, congenital, 165  
     effects of intracranial pressure on, 21  
     examination of projections for, 9  
         technique of, 7  
     fractures of, 178, 368  
         depressed, 181  
         differential diagnosis of, 369  
         linear 180  
         punctured or penetrating, 182  
         repair of, 183  
         technique of examination of, 182  
     hamangioma of 604  
     increased density of, 36  
     infections of, 174  
     local effects of intracranial tumours on, 25  
     localised thinning of, causes of, 34  
     malformations of, congenital and infantile,  
         165 343  
     necrosis of due to X rays or radium, 175  
     neoplasm of, diagnosis from xanthomatosis,  
         539  
     osteitis of 174  
     osteoma of 38  
     osteomyelitis of 411  
     radiographic examination of, general tech  
         nique and pathology, 3  
     radiological features of, normal and patho  
         logical, 30  
     reaction of to invasive tumours, 26  
         to non invasive tumours, 27  
     sarcoma of 38  
     size and shape of, 30  
     suture lines of, diagnosis from fracture, 369  
         tam o shanter, 169 521, 527  
     thickening of, 37  
     thinning of conditions associated with, 40  
     tumours of, carcinoma, 177  
         by direct spread 178  
         cholesteatoma, 177  
         chordoma, 177  
         haemangioma 176  
         metastatic, 177  
         myeloma, 177  
         osteoma, 176  
         sarcoma, 177  
     vascular channels of, 40  
     vault of, normal, 31  
         convolutional markings of, 32  
         localised areas of thinning in, 32  
         porosity of, 32  
         pathological, diminished density, 34  
         increased density, 36  
     venous channels of, diagnosis from fractures,  
         369

- Smith Petersen pin, insertion of, radiological appearances of, 508
- Sphenoid lesser wing of, normal and pathological appearances of 56 57
- Spider fingers, 360
- Spina bifida, 354
- occulta 354
- ventosa 415
- Spinal canal, tumours arising within, 189
- cord anatomy of, 186
- lesions of, soft tissue ossification in 658
- pressure on diseases causing, 189
- tumours of, 185
- diagnosis of by air or by oxygen in injections, 197
- by intrathecal lipiodol, 191
- indications, 191
- interpretation of results, 195
- technique, cisternal injection, 193
- lumbar injection 194
- extradural, 190
- extramedullary, 190
- intramedullary, 191
- radiographic examination technique, 185
- radiological features of, 186
- Spine, carcinoma of, secondary, 189
- fractures of 380
- diagnosis from malacia, 388
- from neoplasm, 388
- from tuberculosis, 388
- laminae 386
- site of, incidence of, 386
- spinous and transverse processes, 386
- vertebrae 386
- fusion of, Albee's and Hibbs methods, 512
- in adult variations in, 297
- X ray appearances in, 296
- in children, lateral view of 295
- variations in, 295
- malformations of congenital 350
- cervical region 351
- dorsal region, 352
- lumbar and sacral regions, 353
- normal, 293
- operations on, Albee's, 512
- Hibbs's, 512
- ossification of, 293
- osteochondritis of, primary and secondary centres of 474
- osteomyelitis of, 412
- radiological features of, 186
- tuberculosis of, abscess formation in, 420
- age and sex incidence of 419
- types of, 419
- tumours of, 188
- malignant, 189
- Splanchnomegaly, 130
- Spondylitis ankylopoietica, 470
- diagnosis from tuberculosis of sacro iliac joint, 431
- from tuberculosis of shoulder 429
- intervertebral discs in 503
- Bechterew type, 471
- chronic, 470
- Spondylitis infective, chronic, 470
- Marie Strumpell type, 471
- muscularis, 472
- ossificans ligamentosa, 471
- radiographic appearances of, 472
- osteo arthritica 470
- rheumatoid, 470
- Spondylöse heredo traumaticque, 471
- rhizo meliques, 471
- Sporotrichosis in bones, 455
- Sprengel's deformity, 356
- Stand upright radiographic, Graham Hodgson, 210
- Steatorrhoea idiopathic 528
- skull changes in, 172
- Steeple head, 166
- Stereoscopy in examination of bones and joints, 251
- in localisation of foreign bodies, 675
- value of, in intracranial examination, 8
- Sterno clavicular joint, dislocation of, 370
- Still's disease 461
- diagnosis from osteochondritis of femoral head epiphysis, 484
- Stomach, filling of, examination of by cine radiography, 779
- normal, effect of delay in emptying, examination by cineradiography, 780
- movements of, examination of by cine radiography 779
- Subarachnoid space anatomy of, 76
- Subastragaloid joints, tuberculosis of, 431
- Subdural calcification, 64
- Suprarenal body, neoplasms of, 555
- Sweet's method and apparatus for localisation of foreign bodies in eye, 748-755
- Sylvius, aqueduct of, anatomy of, 76
- Syndactyly, 359 361
- Synostosis of bones of forearm, 358
- Syphilis, congenital, bone changes in, diagnosis from osteopetrosis 570
- diagnosis from scurvy, 533
- tarda, 440 See also Bone, syphilis of
- tertiary, aseptic bone necrosis in 329
- Syringomyelia, bone changes in, 497
- Charcot's joints in 448
- Syringomyelocoele, 354

## T

- Tabes bone changes in, 497
- Tarsus, examination of, 273
- joint cavities of, 280
- operations on, arthrodesis, 508
- astragalectomy, 510
- Lambrinudi's 510
- Naughton Dunn's 509
- ossification of 276
- osteomyelitis of, 413
- tuberculosis of, 431
- Tear ducts, radiological appearances of, 758
- Teeth and jaws, normal, 681
- in children, 688
- caries of, radiological appearances of, 705
- cysts of, 719

- Teeth, dead, radiological appearances of, 707  
 dilacerated, 694  
 filling materials in, radiological appearances of, 707  
 fractured, 716  
 geminated, 694  
 normal, radiogram of, 681  
 pulp changes in, 707  
 granuloma of, 709  
 root absorption of, 709  
 roots of, buried, radiological appearances of, 710  
 enamel nodule of, 725  
 perforation by instrumentation radiological appearances of, 718  
 variations in, 698  
 supernumerary, radiograms of, 699  
 supplemental, 694  
 tartar on, 711  
 unerupted, infection of follicle of, results of, 721, 722  
 radiograms of, 690
- Temporal bones, anatomy of, mastoid process, 232  
 petrous bone, 230  
 squamous portion, 230  
 lobe tumours, angiography in, 123
- Temporo mandibular joint, ankylosis of 738  
 arthritis of, 738  
 dislocation of, 739  
 injuries to meniscus of, 738
- Tendinitis, calcareous, 649
- Tendons, calcareous deposits in, 649  
 ossification in, 650
- Tenon's capsule, injection of contrast media into, for eye examination, 745, 747, 758
- Teratomas, intracranial, 148
- Thorax, examination of, by cineradiography, 775
- Thorotrast, use of, in cerebral angiography, 116
- Thumb, phalanges of, normal appearances of, 255
- Thyroglossal fistula, 668
- Tibia, head of, fractures of, 380  
 ossification of, 286  
 lower end, examination of, standard views for, 281  
 ossification of, 292  
 malformations of, congenital, 301  
 platyemic, 282  
 shaft of, examination of, 282  
 tubercle of, anatomical development of, 488  
 osteochondritis of, 488  
 upper end of, examination of, 284
- Tissues, reaction of, to dead bone, 325  
 soft, calcification in, due to parasites, 650  
 dystrophic, 646  
 general considerations, 644  
 metastatic or metabolic, 647  
 radiological appearances of, 647  
 fat in, 665  
 gas in, causes of, 662  
 opacities in, due to therapeutic injections, 667
- Tissues, soft, ossification in, 655  
 general considerations, 644  
 in spinal cord lesions, 658
- Tomography of skull, 21
- Trapezium and first metacarpal joint space, 200  
 normal appearances of, 256
- Trapezoid, normal appearance of, 256
- Triangulation methods in localisation of foreign bodies, 675
- Trichina spiralis*, calcification of, 652
- Trigonocephaly, 165
- Trochanters, great and small, epiphyses of, ossification of, 291
- Trochlear epiphysis, ossification of, 265
- Trummerfeldzone in scurvy, 531
- Tuberculoma, calcification in, 67  
 intracranial, 151
- Tuberculosis of bone, 414
- Turnours, cerebellar, ventriculography in, 110  
 frontal, ventriculography in 99  
 intracranial *See also* Intracranial tumours  
 intrasellar, enlargement of sella in, 48  
 occipital, ventriculography in, 103  
 of corpus callosum and septum lucidum, ventriculography in 106  
 of the basal ganglia and caudate nucleus, ventriculography in, 106  
 of third ventricle and midbrain, ventriculography in, 107  
 parietal, ventriculography in, 101  
 pontine ventriculography in, 111  
 temporal ventriculography in, 104  
 sella and suprasellar epidermoid, 132
- Turnschadel 166
- U
- Ulna and radius shafts, examination of, standard positions for, 262  
 fractures of, 374  
 lower end of, normal appearance of, 258  
 ossification of, 262  
 malformations of congenital, 357  
 upper end of, examination of 264
- Uniform normal appearance of, 256
- Undulant fever bone lesions in, 452
- V
- Veins, calcification in, 647  
 cerebral, normal appearances of, 41  
 meningeal, normal appearances of, 42  
 pathological appearances of, 41
- Velter's method of localisation of foreign bodies in eye, 757
- Ventricles, dilated, X ray appearance of, significance of, 90-93  
 displacement of X ray appearance of, 96  
 fourth, anatomy of, 76  
 lateral, anatomy of, 74  
 anterior horn of, 74  
 body of, 75  
 descending horn of, 75  
 posterior horn of, 75  
 narrowing or obliteration of, X ray appearances of, 93-96



- Ventricle, third, anatomy of, 75  
 colloid cyst of, 146  
 dilatation of, effect on sella turcica, 49, 51  
 tumours of, ventriculography in, 107
- Ventricular estimation, 87
- system, changes in, in tumours and other expanding lesions, 89  
 displacement of, 96  
 narrowing or obliteration of, 93  
 normal radiological appearances of, 77
- Ventriculogram, antero posterior projection, 78  
 normal lateral projection, 77, 78  
 occipito frontal projection, 79  
 of arachnoiditis, 111  
 of basal ganglia and caudate nucleus tumours, 106  
 of cerebellar tumours, 110  
 of corpus callosum and septum lucidum tumours, 106  
 of dilated ventricles, 90-93  
 of displacement of ventricle, 96-99  
 of epidermoid tumours, 134  
 of frontal tumours, 99  
 of narrowing or obliteration of ventricle, 93-95  
 of occipital tumours, 103  
 of parietal tumours, 101  
 of pontine tumours, 111  
 of scarring and atrophic lesions of brain, 112  
 of temporal tumours, 104  
 of third ventricle and midbrain tumours, 107  
 postero anterior projection, 79
- Ventriculography, 71  
 contraindications of, 86  
 dangers of, 86  
 in brain abscess, 159  
 in colloid cyst of third ventricle, 147  
 in subdural hæmatoma, 158  
 in tumours and other expanding lesions, 89  
 technique of antero posterior projection, 83  
 injection of air, 81  
 lateral projection, 84  
 postero anterior projection, 83  
 radiographic projections, 82  
 positions of patient, 85
- Vertebra, abnormalities of, 350-351, 353  
 expanding lesions of, 188  
 fifth lumbar, sacralisation of, 353  
 fractures of, 386  
 giant cell tumours of, 188  
 hæmangioma of, 188-603  
 in adult, 296  
 variations of, 297  
 in child, 295  
 ossification of, 293  
 solitary dense, 388  
 wedged, 387, 393

- Vertebral column, normal, 293  
 epiphyseal plates, normal development of, 477  
 osteochondritis of, 477
- Vestibule, anatomy of, 232
- Vitamin C deficiency in etiology of scurvy, 53
- Vitamin D deficiency in etiology of osteomalacia, 524, 525  
 in etiology of rickets, 522, 524, 525  
 treatment of osteomalacia, 526  
 of rickets, 522
- Vitreous, ossification of, 745
- Vogt's method of localisation of foreign bodies in eye, 757
- Volkmann's congenital ankle deformity, 366
- Voorhoeve's disease, 517

## W

- Ward's triangle, 289
- Wessely's method for localisation of foreign bodies in eye, 757
- Whitman's astragalectomy, 510
- Wimberger's sign in scurvy, 532
- Wrist, accessory ossicles of, 259  
 and hand, examination of standard positions, 255  
 appearance of, in full radial flexion, 261  
 in full ulnar flexion, 261  
 with hand in normal position, 261  
 dislocation of, 376  
 fractures of, 375  
 joint cavities in, 260  
 movements of, 261  
 ossification of, 261  
 rheumatic diseases of (diagnosis from tuberculosis), 426  
 tuberculosis of, 426  
 differential diagnosis of, 426

## X

- Xanthomatosis, clinical features of, 537  
 differential diagnosis of, 539  
 radiological appearances of bones in, 538  
 skull changes in, 40-172  
 treatment of, 540

## Y

- Yaws, bone lesions in, 450  
 diagnosis from syphilitic dactylitis, 440

## Z

- Zygoma, fractures of, 400